

Molecular Mimicry: Infection Inducing Autoimmune Disease (Current Topics in Microbiology and Immunology)

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The purpose of this review is to examine the potential role of molecular mimicry in the pathogenesis of human T-lymphotropic virus type 1 ((HTLV- 1)-associated myelopathy/tropical spastic paraparesis (HAM/TSP)). Comp- hensive reviews on the pathogenic mechanisms of HTLV-1-associated human diseases are available throughout the medical literature (Bangham 2000,, 2003; Barmak et al. 2003; Jacobson 2002; Levin and Jacobson 1997; Nagai and Osame 2003; Osame 2002). Approximately 25 years ago the ?rst human retrovirus, HTLV-1, was isolated (Poeisz et al. 1980). Subsequently, infection with HTLV-1 was shown to cause adult T-cell leukemia (ATL) and HAM/TSP (Gessain et al. 1985; McFarlin and Blattner 1991; Osame et al. 1986; Poeisz et al. 1980; Yoshida et al. 1987). HTLV-1 may infect up to 30% of people in endemic areas and 10-20 million people worldwide (Barmak et al. 2003; Edlich et al. 2000). However, only 1%-5% develop either ATL or HAM/TSP, the remainder being clinically asymptomatic carriers of HTLV-1 (Bangham 2000, 2003; Barmak et al. 2003; Jacobson 2002; Levin and Jacobson 1997; Nagai and Osame 2003; Osame 2002). Why infection with HTLV-1 causes ATL or HAM/TSP in some people while the vast majority of individuals are asymptomatic is largely - known. Some possible factors that may differentiate the asymptomatic from the diseased state include viral strain, human histocompatibility leukocyte antigen (HLA), viral load, and the immune response (Bangham 2000, 2003; Barmak et al. 2003; Jacobson 2002; Levin and Jacobson 1997; Nagai and Osame 2003; Nagai et al. 1998; Niewiesk et al. 1994; Osame 2002).

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