

Dynamics and cell-to-cell spread of HTLV-1 infection

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The human T-lymphotropic virus type 1 (HTLV-1) induces a vigorous T-cell response, and the infection results in inflammatory diseases, such as HTLV-1-associated myelopathy/tropical spastic paraparesis (HAM/TSP) in 1% to 5% of infected individuals. The proviral load of HTLV-1 reaches an equilibrium “set point” in each infected person, but the load can vary by more than 10,000-fold between individuals. A high proviral load is associated with a high risk of HAM/TSP. However, it has been widely believed that HTLV-1 is a latent virus, and it is not understood how the proviral load is maintained.

We aim to answer two main questions: first, what determines an individual’s proviral load? Second, why do some HTLV-1-infected people develop HAM/TSP, whereas the majority remain asymptomatic carriers? I shall summarize evidence from cellular immunology, population genetics, DNA microarrays and cell biology for two main conclusions: 1) HTLV-1 is not latent, but persistently attempts to replicate; and 2) the genetically determined efficiency of the HTLV-1-specific cytotoxic T-lymphocyte response is a major determinant of the proviral load and (therefore) of the risk of HAM/TSP.

We now aim to define the “efficiency” of the anti-HTLV-1 immune response in terms of T-cell

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LT20

Hosted by Prof Hew Choy Leong

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