



MEETING ABSTRACT

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The plasticity of HTLV-1 infected CD4+CD25+CCR4+ T-cells through HTLV-1 tax in HAM/TSP

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Recently, it has become increasingly clear that some committed effector and regulatory T (Treg) cells are not stable, and the plasticity of committed T-cells may be related to autoimmunity and inflammatory disease. However, the environmental (extrinsic) molecules that allow for plasticity have not yet been clearly understood.

In Human T-lymphotropic virus type 1 (HTLV-1) associated myelopathy /tropical spastic paraparesis (HAM/TSP), the pathogenesis is known as HTLV-1 infected CD4+ T-cells triggered hyper immune response, which leads to chronic inflammation of the central nervous system. In our previous study, we demonstrated that the majority of CD4+CD25+CCR4+ T-cells were infected with HTLV-1 and that this T-cell subset was increased in HAM/TSP. Although CD4+CD25+CCR4+ T-cells of healthy condition include suppressive T cell subsets such as Treg and Th2, this T-cell subset becomes Th1-like cells with overproduction of IFN- γ in HAM/TSP patients (PLoS ONE 2009). Since HTLV-1 tax is known to up-regulate the expression of several proinflammatory cytokines, and importantly, the level of HTLV-1 tax mRNA expression is reported to correlate with disease severity in HAM/TSP patients, we hypothesize that HTLV-1 tax may convert HTLV-1 infected T-cells into abnormal IFN- γ producing Th1-like T-cells in HAM/TSP. In this study, we present the molecular mechanisms underlying the plasticity of HTLV-1 infected CD4+CD25+CCR4+ T-cells through HTLV-1 tax.

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