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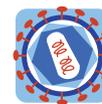
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MEETING ABSTRACT

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An interferon signature is associated with HAM/TSP but not viral containment in HTLV-1 infection

Sonja Tattermusch^{1*}, Jason Skinner², Damien Chaussabel², Jacques Banchereau², Matthew P Berry³, Anne O'Garra³, Graham P Taylor¹, Charles R M Bangham¹

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Most people infected with Human T-cell Lymphotropic Virus Type 1 (HTLV-1) remain clinically asymptomatic; however, a minority develops the debilitating myelopathy HAM/TSP. Current treatment of HAM/TSP is limited by our partial understanding of the protective immune response to HTLV-1 and the pathogenesis of HAM/TSP.

We wished to test the hypothesis that a gene expression signature in peripheral blood distinguishes between patients with HAM/TSP and ACs. We investigated genome-wide transcription patterns in whole blood from HTLV-1 asymptomatic carriers (AC; n=37), patients with HAM/TSP (n=20) and uninfected control subjects (n=17). We identified a 542-gene signature that was deregulated in all HTLV-1+ individuals and predominantly comprised transcripts involved in p53-mediated DNA damage responses (p=0.00489). An 80-gene signature distinguished patients with HAM/TSP from those with the clinically similar disease multiple sclerosis. Paradoxically, at a given proviral load patients with HAM/TSP, but not ACs, over-expressed antiviral interferon-stimulated genes (ISGs; p=0.00859).

Expression of these ISGs (assessed by quantitative PCR and flow cytometry) was not limited to HTLV-1-infected CD4+ T cells, suggesting that all peripheral blood immune cells were exposed to interferons (IFN) *in vivo*. Neither elevated IFN plasma levels nor an abnormal capacity for IFN production was detected in patients with HAM/TSP. However, peripheral immune cells in patients with HAM/TSP were more sensitive to IFN-alpha and IFN-gamma stimulation.

These findings suggest that chronic over-expression of a specific subset of ISGs is ineffective in containing HTLV-1 and may instead contribute to the pathogenesis of HTLV-1-associated myelopathy.

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Author details

¹Departments of Immunology and GU Medicine and Communicable Diseases, Imperial College London, London, W2 1PG, UK. ²Baylor Institute for Immunology Research, INSERM U-899, Dallas, TX 75204, USA. ³Division of Immunoregulation, MRC National Institute for Medical Research, London, NW7 1AA, UK.

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* Correspondence: sonja.tattermusch07@imperial.ac.uk

¹Departments of Immunology and GU Medicine and Communicable Diseases, Imperial College London, London, W2 1PG, UK

Full list of author information is available at the end of the article