

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

Sonja Tattermusch, Jason Skinner, Damien Chaussabel, Jacques Banchereau,
Matthew Berry, Anne O'Garra, Graham Taylor, Charles Bangham

► **To cite this version:**

Sonja Tattermusch, Jason Skinner, Damien Chaussabel, Jacques Banchereau, Matthew Berry, et al..
An interferon signature is associated with HAM/TSP but not viral containment in HTLV-1 infection.
15th International Conference on Human Retroviruses: HTLV and Related Viruses, Jun 2011, Leuven
and Gembloux, Belgium. pp.A108, 10.1186/1742-4690-8-S1-A108 . inserm-00663943

HAL Id: inserm-00663943

<https://www.hal.inserm.fr/inserm-00663943>

Submitted on 27 Jan 2012

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.



MEETING ABSTRACT

Open Access

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

From 15th International Conference on Human Retroviruses: HTLV and Related Viruses Leuven and Gembloux, Belgium. 5-8 June 2011

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.

Acknowledgements

This study is funded by the Wellcome Trust (UK).

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.

Published: 6 June 2011

doi:10.1186/1742-4690-8-S1-A108

Cite this article as: Tattermusch *et al.*: An interferon signature is associated with HAM/TSP but not viral containment in HTLV-1 infection. *Retrovirology* 2011 **8**(Suppl 1):A108.

Submit your next manuscript to BioMed Central and take full advantage of:

- Convenient online submission
- Thorough peer review
- No space constraints or color figure charges
- Immediate publication on acceptance
- Inclusion in PubMed, CAS, Scopus and Google Scholar
- Research which is freely available for redistribution

Submit your manuscript at
www.biomedcentral.com/submit



* 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