



POSTER PRESENTATION

Open Access

HTLV-1 infected CD4+T cells of HAM/TSP patients are susceptible to tunicamycin-induced endoplasmic reticulum stress

Daisuke Kodama^{1*}, Ryuji Kubota¹, Toshio Matsuzaki², Hiroshi Takashima³, Shuji Izumo¹

From 17th International Conference on Human Retroviruses: HTLV and Related Viruses
Trois Ilets, Martinique. 18-21 June 2015

Lectin array analysis for membrane proteins from CD4+T cells of four respective cases of HAM, healthy carriers (HC), and negative controls (NC) showed significant high signal for *Urtica dioica* agglutinin (UDA) and *Solanum tuberosum* (Potato) (STL) lectins, that specifically recognize N-glycan, N-acetyllactosamine (LacNAc) which consist of repeats of the disaccharide $\beta\text{Gal}(1-4)\beta\text{GlcNAc}(1-3)$. We verified that LacNAc is significantly expressed in CD4+T cells in HAM by lectin ELISA for each ten cases of HAM, HC, and NC, followed by qRT-PCR of $\beta 1,3$ -N-acetylglucosaminyl-transferase 2 (B3GNT2) gene which was reported as main responsible enzyme in LacNAc biosynthesis. Using tunicamycin(TM), known as a N-glycan inhibitor and an apoptosis inducing agent through endoplasmic reticulum (ER) stress, we investigated the effect of LacNAc suppression on CD4+T cells. In NC, both of LacNAc+ and LacNAc-cells showed similar survival regardless of treatment with TM of 2 μM for 24h or without TM, however, both cells in HAM showed worse survival than in NC and LacNAc+cells showed better survival when treated with TM. Moreover in HAM, Tax-cells showed better survival regardless of treatment with TM and LacNAc+cells when with treatment with TM survived significantly better than when without TM. Tax+cells showed worse survival than Tax-cells and tended to decrease when treated with TM. Collectively, we hypothesized that LacNAc is a resistant factor to TM-induced apoptosis in HTLV-1 uninfected cells but HTLV-1 infected cells

may be susceptible to TM even if LacNAc are expressed. To confirm this hypothesis, we performed qRT-PCR of main effector genes in ER stress pathway: x-box binding protein 1, spliced variant (XBP1s), heat shock 70kDa protein 5(GRP78), and heat shock protein 90kDa beta, member 1(GRP94). Only GRP94 was significantly suppressed in HAM and HC. We conclude that HTLV-1 infected cells may be under ER stress and be susceptible to TM, and that LacNAc positive cells are resultant survivors.

Authors' details

¹Molecular Pathology, Center for Chronic Viral Diseases, Kagoshima University Graduate School of Medical and Dental Sciences. Kagoshima-Shi, Kagoshima-Ken, 890-8544, Japan. ²Medical Corporation Sanshukai Ohkatsu Hospital, Kagoshima-Shi, Kagoshima-Ken, 890-67, Japan. ³Department of Neurology, Kagoshima University Graduate School of Medical and Dental Sciences. Kagoshima-Shi, Kagoshima-Ken, 890-8520, Japan.

Published: 28 August 2015

doi:10.1186/1742-4690-12-S1-P19

Cite this article as: Kodama et al.: HTLV-1 infected CD4+T cells of HAM/TSP patients are susceptible to tunicamycin-induced endoplasmic reticulum stress. *Retrovirology* 2015 **12**(Suppl 1):P19.

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: Daisuke.Kodama@mb2.seikyoku.ne.jp

¹Molecular Pathology, Center for Chronic Viral Diseases, Kagoshima University Graduate School of Medical and Dental Sciences. Kagoshima-Shi, Kagoshima-Ken, 890-8544, Japan

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

