



**-HUMAN ENDOGENOUS  
RETROVIRUS TYPE W  
ANTIGENAEMIA (MSRV-  
ENVELOPE) IN PATIENTS  
WITH CIDP.**

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**Body of abstract:**

The human genome comprises nearly 45% of mobile genetic elements, in which human endogenous retroviruses (HERV) represent 8% of total DNA. HERV-W family retains copies expressing an envelope protein (MSRV-Env), which activates a pro-inflammatory and autoimmune cascade through interaction with Toll-Like receptor 4 (TLR4) on antigen-presenting cells. HERV-W has now consistently been associated with Multiple Sclerosis (MS) and HERV-W was shown to be activated by infectious agents suspected to play a role in the ethiopathogeny of MS. Nonetheless, in a pilot study addressing various neurological diseases, a cluster of patients with CIDP also revealed to have positive antigenaemia for MSRV-Env. Since CIDP shares common and analogous features with MS pathogenesis, we have initiated a study exploring whether this immunopathogenic HERV-W protein could be also be involved in certain cases of CIDP.

For the purpose, we have performed ELISA quantification of HERV-W/MSRV-Env antigen from CIDP patients, neurological and healthy controls with a dedicated assay.

The results obtained with present numbers of patients included in the study revealed 15/30 (50%) positives in CIDP from Créteil (France) and 9/19 (47%) from Lausanne (Switzerland),

but only 3/18 (17%) in apparently healthy controls (HC). A statistical difference with controls is reached with Chi-square test for separate CIDP series ( $p < 0.05$ ) or with CIDP cases altogether (24/49,  $p < 0.01$ ). Inclusion of controls with other neurological diseases has revealed few positive (4/20;  $p < 0.02$  compared with all CIDP), comprising one case with spastic paraparesis, two with metabolic neuropathies and one with Parkinson's disease.

The present results confirm the existence of a group (about one-half) of CIDP patients with positive antigenaemia for HERV-W neuroinflammatory envelope protein. The potential involvement of this HERV protein in the pathophysiology of certain cases of CIDP is now raised. Such an involvement of HERV-W in CIDP may open novel and targeted therapeutic perspectives.