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## Profile of long-term non-progressor HIV-1 infected patients in follow-up at a tertiary hospital, Southern Brazil

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In this study, we evaluated the HIV-1 features, the clinical and epidemiological profile of LTNP patients treated in a referral hospital from Curitiba, Paraná, Southern Brazil. Medical records were reviewed and periodic blood draws were held for tests aimed to determine the genetic variability of HIV-1 and to evaluate the host innate and genetic patterns. To date, 23 patients, corresponding to 1.64% of patients followed at the Infectious Diseases Division from HC/UFPR were identified as LTNPs. The gender distribution is homogeneous (56% female) with a median age of 45 years (range 12-62 years). The risk behavior for HIV-1 infection was sexual activity in 65% (73% were heterosexual), vertical infection in 22% and use of injectable drugs in 13% of the cases. The median time of HIV-1 diagnosis was 13.5 years (range 9-23 years) and the median count CD4+ T lymphocytes of 702 cells per mm<sup>3</sup> (range 569-890). Regarding host protective factors, we observed a frequency of protective HLA-B alleles in 20/22 (91%) of analyzed individuals. Deletion of the CCR5 co-receptor gene encoding was investigated in all patients and it was found in four (17.4%). The genotypic HIV-1 evaluation showed subtypes C (52%), B (30%) and recombinants BC and BF (18%). Tropism prediction was consistent with an R5 phenotype in almost all study patients (95%). Several mechanisms, both viral and host, appear to be associated with the status of this Cohort of HIV+ LTNP. Data obtained to date well characterized our group LTNPs studied as described in the literature with profile related to a delayed clinical progression. As most studies on LTNPs and HIV pathogenesis were carried out with subtype B, further research is needed to elucidate the host-viral interactions in individuals with various clades of HIV-1, seeking to clarify the reasons for their slow progress, consequently being important for future therapeutic options for HIV infection.

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## Effect of HIV and HCV infections on interaction of pDCs and NK cells: A population-based study

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Previous studies have shown the interaction between plasmacytoid dendritic cells (pDCs) and nature killer (NK) cells is critical for host innate immunity against viral infections. However, the pDC-NK interaction mechanisms as well as the effects of HIV/HCV infections on this interaction remain to be determined. According to the infection status, participants were enrolled and divided into four groups: HIV-1 mono-infection, HCV mono-infection, HIV/HCV co-infection and healthy control groups. pDCs and NK cells were isolated from PBMCs of the subjects and related cellular factors were measured and compared among different groups. From healthy subjects, the results showed that pDCs produced high level of IFN- $\alpha$  through activation by TLR-9 receptor agonist ODN2216 treatment *ex vivo*; whereas NK cells were not directly activated by ODN2216 treatment but produced cytokines (IFN- $\gamma$ , perforin, granzyme-B) by co-culture with ODN2216-activated pDCs. pDCs from viral infected patients (HIV, HCV, HIV/HCV) exhibited higher levels of IFN- $\alpha$  compared to pDCs from healthy subjects, indicating viral infections could activate pDCs *in vivo*. However, HIV infections (HIV, HIV/HCV) resulted in no response of pDC to ODN2216 treatment *ex vivo*, whereas HCV mono-infection had little effect on pDC activation by ODN2216 treatment. Correspondingly, in HIV and HIV/HCV groups, NK cells were no longer activated by co-culture with ODN2216-treated pDCs, whereas in HCV group, NK cells were still activated by co-culture with ODN2216-activated pDCs. Taken together, our results indicate that HIV infection impairs pDC function and thus disrupts subsequent pDC-NK interaction and HCV infection has little impact on pDC function and pDC-NK interaction.

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