CXCR4 with gp120, can cause upregulation of TRAIL receptors and consequent sensitivity to apoptosis by this pathway. In this work, our aim was to measure if CD4+ T lymphocytes from HIV+ patients, had increased expression of TRAIL ligand and death receptors (DR) 5/4 and coreceptor CXCR4 and, evaluate its association.

**Methods** Ten HIV+ ART naïve patients and seven HIV- donors were recruited. The immunophenotyping of CD4+ T lymphocytes was performed with CD3+/CD4+ labeling, after that the TRAIL death receptors DR4 and DR5 as well as the TRAIL ligand and the coreceptor CXCR4 were measured for expression and MFI (median fluorescence intensity) by flow cytometry in whole blood samples.

**Results** CD4+T lymphocytes from HIV+ patients showed an augmented expression and MFI of both death receptors and TRAIL ligand compared with the healthy controls; on the other hand, expression of coreceptor CXCR4 was increased in the HIV+ group and the MFI showed a significant difference compared to healthy subjects. Correlations between DR4 and the TRAIL ligand with CXCR4 showed no significance; in contrast, both expression and MFI of DR5 and CXCR5 were significant and showed a strong direct correlation (expression: p<0.05 r=-0.69 and MFI: p<0.005 r=0.81).

**Conclusion** There is evidence of apoptosis triggered by CXCR4 activation, not related to the Fas pathway, which is one of the main causes related to cell depletion in HIV infection. The upregulation of the TRAIL pathway in HIV infected subjects is correlated with the CXCR4 expression, which could be the cause of the reported apoptosis in these patients.

**Disclosure** No significant relationships.

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**EFAVIRENZ BASED ANTI-RETROVIRAL REGIMENS IN PAEDIATRICS: A PEEP INTO NEUROPSYCHIATRIC AND BIOCHEMICAL DERANGEMENTS**

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**Background** Chronic inflammation in HIV infection has been associated with accelerated disease progression. Several external host factors contribute to inflammation like, microbial translocation and coinfections; as well as host intrinsic mechanisms; such as immune activation and the regulation of factors that participate in inflammation pathways. Members of the transcriptional factors ZBTB have been associated with repression of proinflammatory cytokines; the objective of this work was to identify if ZTB B repressors could be contributing to cytokines levels decrease in HIV+ ART-treated patients.

**Methods** CD4+ T cells were isolated from 12 HIV- and 21 HIV++, subclassified as naïve (n= 8) and treated (undetectable for >1 year, n = 13). Gene expression of ZBTB2, ZBTB4, ZBTB7B, ZBTB17, ZBTB38, BCL6 and ZNF131 was determined by qRT-PCR. Cytokine levels were quantified (IL-1β, IFN-α, IFN-γ, TNF-α, MCP-1, IL-6, IL-8, IL-10, IL-12, IL-17, IL-18, IL-23, IL-33) by flow cytometry in serum. Kruskal-Wallis test was used for statistical analysis.

**Results** In naïve patients, only ZBTB17 showed a significant up-regulation (2.76 fold). BCL6, ZBTB4, ZBTB38 had an increase but, were not significant (1.01, 0.91, 0.13 fold respectively); treated patients showed a significant up-regulation in ZBTB17, ZBTB4 and ZBTB38 (2.23, 1.13, 1.04 fold respectively) and, BCL6 had 1.02 fold, however, it was no...