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Could *NLRP-3* exert a distinct role in HIV-1 pathogenesis depending on the cell type?

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HIV+ patients are characterized by a chronic inflammation, in part mediated by IL-1β constitutive release. Inflammasome assembly leads to the activation of caspase-1 and consequent cleavage and release of IL-1β and IL-18. NLRP3-inflammasome is responsible for HIV-1 detection in monocytes and dendritic cells, suggesting that it could be involved in the first steps of HIV-1 infection, and in the establishment of chronic inflammation in infected subjects. Our group demonstrated that the gain-of-function 3'UTR variant rs10754558 in NLRP3 gene confers protection against HIV-1 infection, in part corroborating this hypothesis. On the other hand, it was recently shown that in T CD4+ lymphocytes *NLRP-3* could act as a transcription co-factor for IL4 transcription leading to a Th2 polarization. Taking in account that a Th2 increase has been associated with bad prognosis of HIV+ individuals and with progression to AIDS, we hypothesize that *NLRP-3* could contribute to HIV pathogenesis not only due to its role in myeloid cells but also in lymphocytes. As the role of *NLRP-3* is poorly elucidated in lymphocytes, aim of this study was to evaluate *NLRP-3* differential expression, *NLRP-3* inflammasome activation and IL-4 induction in distinct lymphocyte populations from healthy (HIV-) individuals as well as in HIV+ patients. *NLRP-3* activity in lymphocytes was then correlated with the differential distribution of inflammasome SNPs in HIV+ cohort. Our results suggested that *NLRP-3* could exert a role in lymphocytes dysregulation in HIV+ patients.

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