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Myeloid cells are key targets of human immunodeficiency virus (HIV) replication *in vivo* and are important players in viral-induced pathogenesis. Albeit with variations depending on their differentiation status, these cells are less susceptible to viral infection than other cell types like primary lymphocytes and established cell lines *ex vivo*. This resistance is obviously exerted at multiple levels, yet a major restriction during lentiviral infection occurs during the early steps of infection, that is, during those steps comprised between viral entry and viral DNA integration.

To date, a single viral non structural protein has been identified as capable of removing this restriction in primary myeloid cells, the Vpx protein. Vpx is coded by members of the SIV$_{SM}$/HIV-2 lineage, but is absent in HIV-1 and in most of the remaining SIV lineages. We have already shown that Vpx is not only required for the completion of infection by the parental HIV-2 and SIV$_{SM}$ viruses, but also that Vpx exerts a positive effect during the infection of myeloid cells with a number of distantly related lentiviruses, like HIV-1, FIV and EIAV. In all these cases, Vpx promoted the accumulation of complete viral DNA and this accumulation correlated with an increased efficacy of infection.

The effect of Vpx has been linked to the action of an E3 ubiquitin ligase complex, but this action remains controversial and the mechanism and the effects through which it may be exerted are unclear. In an effort to understand the mechanistic role of Vpx in the removal of the restriction to lentiviral infection existing in myeloid cells, we have performed a wide analysis of the post-translational modifications that over the years have been described for Vpx, ubiquitination, phosphorylation, and so forth and we have tried to correlate these properties to the functionality of Vpx proteins during the early steps of infection. These and other results will be presented here.

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