

Precursor Forms of Vitamin D Reduce HIV-1 Infection In Vitro

JAIDS Journal of Acquired Immune Deficiency Syndromes. 73(5):497–506, DEC 2016

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DOI: 10.1097/QAI.0000000000001150

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Abstract

Background:

Although the anti-HIV-1 effects of vitamin D (VitD) have been reported, mechanisms behind such protection remain largely unexplored.

Methods:

The effects of two precursor forms (cholecalciferol/calcitriol at 0.01, 1 and 100 nM and calcitriol at 100 and 250 nM) on HIV-1 infection, immune activation, and gene expression were analyzed in vitro in cells of Colombian and Italian healthy donors. We quantified levels of released p24 by enzyme-linked immunosorbent assay, of intracellular p24 and cell-surface expression of CD38 and HLA-DR by flow cytometry, and mRNA expression of antiviral and immunoregulatory genes by real-time reverse transcription-polymerase chain reaction.

Results:

Cholecalciferol decreased the frequency of HIV-1-infected p24+CD4+ T cells and levels of p24 in supernatants in a dose-dependent manner. Moreover, the CD4+CD38+HLA-DR+ and CD4+CD38-HLA-DR+ subpopulations were more susceptible to infection but displayed the greatest cholecalciferol-induced decreases in infection rate by an X4-tropic strain. Likewise, cholecalciferol at its highest concentration decreased the frequency of CD38-HLA-DR+ but not of CD38+HLA-DR+ T-cell subsets. Analyzing the effects of calcitriol, the main VitD source for immune cells and an R5-tropic strain as the most frequently transmitted virus, a reduction in HIV-1 productive infection was also observed. In addition, an increase in mRNA expression of APOBEC3G and PI3 and a reduction of TRIM22 and CCR5 expression, this latter positively correlated with p24 levels, was noted.

Conclusions:

VitD reduces HIV-1 infection in T cells possibly by inducing antiviral gene expression, reducing the viral co-receptor CCR5 and, at least at the highest cholecalciferol concentration, by promoting an HIV-1-restrictive CD38+HLA-DR- immunophenotype.

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Nephrology, dialysis, transplantation : official publication of the European Dialysis and Transplant Association - European Renal Association 2016; 31(5): 698-705.

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