

#### In Vitro Effect of Ascorbic Acid in HIV-1 Infected CD4+ T-cells

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#### Abstract

Human immunodeficiency virus-1 (HIV-1) attacks the immune system by infecting and depleting CD4+ Tcells. HIV infection induces cellular oxidative stress potentially leading to DNA damage. Although no cure has been found for HIV-1, antiretroviral treatment (ART) can control its viral load. Since impoverished countries have limited access to ART, complementary, inexpensive treatments need to be further studied. Studies have shown that micronutrients have a positive effect in limiting HIV-1 pathogenesis. Ascorbic acid (vitamin C) is a known antioxidant micronutrient found in fruits, vegetables and as a dietary supplement. This study aims to assess the effect of ascorbic acid in HIV-1 infected CD4+ T-cells in terms of viral load reduction and protection from oxidative stress-induced DNA damage. We hypothesize that, upon exposure to ascorbic acid, there will be a reduction in viral load and oxidative stress-induced DNA damage in infected CD4+ T-cells. The devised method consists of exposing HIV-1 infected and noninfected CD4+ T-cells to ascorbic acid at concentrations ranging from 2 to 140 µg/ml. At 24, 48 and 72 hours of incubation viral load was measured through gRT-PCR; cell number and viability were assessed with trypan blue dying using an AutoT4 Cellometer and DNA damage was measured at 72 hours through the Comet assay. Comparing the oxidative stress levels and viral counts of HIV-1 infected CD4+ T-cells upon exposure to ascorbic acid can provide a significant insight into the use of this antioxidant in HIV-1 infected CD4+ T-cells in limiting its viral replication.

### Keywords

HIV-1 (Human Immunodeficiency Virus-1); Ascorbic acid; CD4+ cells; Micronutrients

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#### **ABSTRACT**

Human immunodeficiency virus-1 (HIV-1) attacks the immune system by infecting and depleting CD4+ T-cells. HIV infection induces cellular oxidative stress potentially leading to DNA damage. Although no cure has been found for HIV-1, antiretroviral treatment (ART) can control its viral load. Since impoverished countries have limited access to ART, complementary, inexpensive treatments need to be further studied. Studies have shown that micronutrients have a positive effect in limiting HIV-1 pathogenesis. Ascorbic acid (vitamin C) is a known antioxidant micronutrient found in fruits, vegetables and as a dietary supplement. This study aims to assess the effect of ascorbic acid in HIV-1 infected CD4+ T-cells in terms of viral load reduction and protection from oxidative stress-induced DNA damage. We hypothesize that, upon exposure to ascorbic acid, there will be a reduction in viral load and oxidative stress-induced DNA damage in infected CD4+ T-cells. The devised method consists of exposing HIV-1 infected and noninfected CD4+ T-cells to ascorbic acid at concentrations ranging from 2 to 140 µg/ml. At 24, 48 and 72 hours of incubation viral load was measured through qRT-PCR; cell number and viability were assessed with trypan blue dying using an AutoT4 Cellometer and DNA damage was measured at 72 hours through the Comet assay. Comparing the oxidative stress levels and viral counts of HIV-1 infected CD4+ T-cells upon exposure to ascorbic acid can provide a significant insight into the use of this antioxidant in HIV-1 infected CD4+ T-cells in limiting its viral replication.

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