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HIV-1 Infection And Endothelial Progenitor Cell Dysfunction

HIV-1 infection is now recognized to be associated with increased cardiovascular morbidity and mortality. The mechanisms responsible for the increased cardiovascular risk within HIV-1 infection are not clear. Endothelial injury without a sufficient compensatory reparative response is thought to accelerate the progression of cardiovascular disease and its clinical consequences, including death. Bone marrow-derived circulating endothelial progenitor cells (EPCs) are now recognized to play a critically important role in maintaining, repairing and/or regenerating the endothelial monolayer and restoring functional activity. EPC dysregulation has been linked to vascular dysfunction, increased atherosclerotic disease risk and greater cardiovascular morbidity and mortality. However, it is currently unknown whether HIV-1-infection per se is associated with diminished EPC function. The aim of this small pilot study was to test the hypothesis that circulating EPC clonogenic and migratory capacity are reduced in HIV-1-infected treatment naïve individuals. Peripheral blood samples were collected from 7 healthy (age: 36 ± 3 yr; BMI 25.4 ± 0.3 kg/m²) and 7 HIV-1-infected (36 ± 2 yr; BMI 24.7 ± 0.8 kg/m²) men. Cells with phenotypic EPC characteristics were isolated from peripheral blood mononuclear cells and EPC clonogenic (colony forming unit [CFU] assay) and migratory (Boyden chamber) capacity were determined. EPC colony-forming capacity was 96% lower in the HIV-1 compared with healthy men. In addition, EPC migration was 36% lower in the HIV-1 compared with healthy men. These very preliminary results suggest that: 1) EPC clonogenic and migratory capacity are lower in HIV-1-infected adults; and 2) EPC dysfunction may contribute to HIV-1-related cardiovascular risk.