**HIV-1 INFECTION IS ASSOCIATED WITH IMPAIRED ENDOTHELIAL PROGENITOR CELL FUNCTION**

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HIV-1 infection is associated with vascular endothelial dysfunction and increased risk of cardiovascular disease. Bone marrow-derived circulating endothelial progenitor cells (EPCs) play a critical role in maintenance, repair and regeneration of the vascular endothelium. EPC dysfunction has been linked to increased vascular disease risk. The experimental aim of this study was to determine the influence of HIV-1 infection and antiretroviral therapy (ART) on EPC function and telomere length. To address this aim we studied 40 men: 20 HIV-1-seronegative (age: 42±2 yr); 10 HIV-1-seropositive treatment-naïve (36±2 yr); and 10 HIV-1-seropositive receiving ART (42±3 yr; efavirenz-based regimen). Cells with phenotypic EPC characteristics were isolated from peripheral blood and the following characteristics assessed: migration (Boyden chamber), angiogenic growth factor release (ELISA), apoptosis (active caspase-3) and telomere length (Southern hybridization). Capacity of EPCs to migrate (816±79 vs 1338±167 AU), release granulocyte-colony stimulating factor (G-CSF; 815±169 vs 1483±188 pg/mL) and resist apoptosis (5.2±0.5 vs 3.0±0.3 ng/mL) was significantly blunted in EPCs from HIV-1-seropositive treatment-naïve vs healthy men. Additionally, HIV-infection was associated with marked telomere attrition (7131±638 vs 9145±565 bp; p<0.05). Of note, there were no significant differences in migratory capacity (752±120 AU), G-CSF release (955±182 ng/mL), caspase-3 (4.1±0.7 ng/mL) or telomere length (7479±358 bp) between the seropositive treatment-naïve men and seropositive men receiving ART. In conclusion, HIV-1 infection, regardless of ART status, is associated with diminished migratory capacity and angiogenic growth factor release, increased apoptotic susceptibility and telomere shortening. Impaired EPC function may contribute to HIV-1-related endovascular dysfunction and cardiovascular risk.