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# O311 Pathogenesis of non-AIDS morbidities in HIV disease and implications for management

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As the ability of highly active antiretroviral therapy (HAART) to suppress virus in a durable and safe manner improves, the inability of HAART to fully restore a normal immune system may emerge as the primary limitation of therapy. Among individuals who initiate HAART at a CD4 cell count <200 and who exhibit a potent and durable virologic response, only 50% are able to achieve normal peripheral CD4+ T cell within the first 10 years of therapy. The inability to restore CD4+ T cell numbers is predicted and perhaps caused by persistent T cell activation on therapy, which in turn may be due to residual HIV replication, persistent microbial translocation, poorly controlled co-infections and/or other mechanisms. Although long-term treated patients with suppressed virus are at low risk for AIDS-related complications, they remain at high risk for significant non-AIDS morbidity, particularly premature cardiovascular disease. Many of these non-AIDS complications are known to be associated with and perhaps caused by chronic inflammation. These data suggest that persistent inflammation during HAART will emerge as primary factor limiting the long-term effectiveness of therapy.