Highly active antiretroviral therapy can effectively control HIV replication in many infected individuals. Some data suggested that viral decay dynamics may depend on the stages of the viral replication cycle inhibited by different classes of drugs. In this talk, I will use a mathematical model including multiple infection stages to study the effect of various drug classes on the viral load dynamics under treatment. The model will be used to explain the discrepancy of the viral load change observed in patients receiving raltegravir and efavirenz based therapy. I will also introduce a model on the basis of a new mechanism to explain the slow time scale of CD4+ T cell decline during chronic HIV infection. Modeling prediction will be compared with long-term CD4+ T cell data in untreated HIV patients. (Received September 22, 2015)