Mathematical modelling studies of HIV infection in-host typically involve the basic virus of dynamics, a system of three ordinary differential equations that tracks uninfected and infected T-cell populations and the HIV viral load. Much has been learned about HIV and HIV infection dynamics using this model, however, it ignores specific attributes of the immune system and immune response, and makes simplifying assumptions on the underlying biological processes. We study models of HIV infection that extend the basic model to include immune system characteristics. We will present examples that result in Hopf and/or backward bifurcations. Relationships to HIV infection in-host will be discussed. (Received September 10, 2015)