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A bi-stable switch in virus dynamics can explain the differences in disease outcome following SIV infections in rhesus macaques

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Experimental studies have shown that size and infectious-stage of viral inoculum influence disease outcomes in rhesus macaques infected with simian immunodeficiency virus. The possible contribution to disease outcome of antibody developed after transmission and/or present in the inoculum in free or bound form is not understood. In this study, we develop a mathematical model of virus-antibody immune complexes formation and use it to predict their role in transmission and protection. The model exhibits bi-stable dynamics between cleared and persistent states. We fitted it to temporal virus data and estimated parameter values for free virus infectivity rate and antibody's carrying capacity for which the model transitions between virus clearance and persistence when the initial conditions (in particular the immune complexes to free virus ratio) vary. We used these results to make predictions on the minimum virus load in the inoculum leading to persistent infection in the presence and absence of protective antibody responses.