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Modelling mutation in equine infectious anemia virus infection suggests a path to viral clearance with repeated vaccination

Equine infectious anemia virus (EIAV) is a lentivirus similar to HIV that infects horses. Clinical and experimental studies demonstrating immune control of EIAV infection hold promise for efforts to produce an HIV vaccine. Antibody infusions have been shown to block both wild-type and mutant virus infection, but the mutant sometimes escapes. Using these data, we develop a mathematical model that describes the interactions between antibodies and both wild-type and mutant virus populations, in the context of continual virus mutation. The antibody infusions are modelled using impulsive differential equations, a technique that offers insight into repeated vaccination by approximating the time-to-peak by an instantaneous change. We use impulsive theory to determine the maximal vaccination intervals that would be required to reduce the wild-type and mutant virus levels below one particle per horse. We show that seven boosts of the antibody vaccine are sufficient to eradicate both the wild-type and the mutant strains. In the case of a mutant virus infection that is given infusions of antibodies targeting wild-type virus (i.e., simulation of a heterologous infection), seven infusions were likewise sufficient to eradicate infection, based upon the data set. However, if the period between infusions was sufficiently increased, both the wild-type and mutant virus would eventually persist in the form of a periodic orbit. These results suggest a route forward to design antibody-based vaccine strategies to control viruses subject to mutant escape.