Title
Within-host processes to population-level disease dynamics: Foot-and-mouth disease in African buffalo.

Mini-symposium title
Modeling Infection Dynamics Across the Scales

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Abstract
Extremely contagious pathogens are among the most important global public and animal health concerns, because of their high burden of morbidity and mortality, their violent outbreaks, and as potential threats to biosecurity. Understanding the conditions and mechanisms that allow for robust persistence of contagious pathogens in their host populations is thus a fundamental problem in disease ecology. Foot-and-mouth-disease virus (FMDV) is one of the most contagious pathogens known to man, and can infect domestic livestock and numerous cloven-hoofed wildlife species (Coetzer et al. 1994).

In sub-Saharan Africa, wild buffalo (Syncerus caffer) act as a reservoir for FMDV, challenging global eradication and local economies (Thomson et al. 1992).

For the first time, we estimated epidemiologic parameters from experimental data and constructed data-driven transmission models to investigate the dynamics of FMD in buffalo, asking how this highly contagious pathogen achieves long-term endemic persistence in its reservoir host populations. We show that all three southern African FMDV serotypes (SAT1, 2, 3) spread readily among buffalo during primary infection. However, the dynamics of the carrier state, which allows for retention of viable virus after initial infection in some hosts, are variable among serotypes, leading to striking epidemiological variation.

As the first study to systematically explore FMD dynamics in its wildlife reservoir, our results reveal that, even in large buffalo herds, none of the serotypes are likely to persist endemically due to calf-to-calf transmission of acute infection alone. Including transmission from carriers in our models suggests robust endemic persistence of SAT1, and possible persistence of SAT3 in large populations, but not of SAT2, the most common serotype infecting livestock. Additional mechanisms of persistence, such as viral antigenic shift, or loss of acquired immunity may be required for the persistence of SAT2 and SAT3 in buffalo populations.
REFERENCES