Exposure-induced individual-level heterogeneity as a possible cause of silent transmission of vector-borne diseases

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Background

Vector-borne diseases, like bluetongue and West Nile fever, pose a severe threat to humans, animals, or both. During outbreaks, there is a noticeable increase in clinical cases. Between outbreaks clinical cases are sporadic. However, these diseases persist in periods where no clinical cases are observed. A possible explanation for this observation could be the coupling between the number of infectious bites received and the proportion of infections that lead to apparent disease, as observed in several vector-borne diseases[1,2]. Here we hypothesize that an explanation for persistence with low clinical disease can be found in dose-dependent individual-level heterogeneity. Hosts that are exposed to a high number of infectious bites, are more likely to develop a high viremia. Hosts with a high viremia are in turn more likely to develop clinical disease and may infect more vectors, Thereby increasing the chance that other hosts receive multiple infectious bites. At the same time, hosts exposed to a low number of infectious bites might develop a low viremia, infect few vectors, and are unlikely to be detected, making them a cause of silent transmission.

Objective

Investigate how individual-level heterogeneity, driven by the number of infectious bites received, can contribute to silent transmission of vector-borne disease.

Methods

A compartmental model was created where hosts and vectors are separated based on the number of infectious bites they were exposed to. The probability of infecting a vector upon a successful bite and the duration of the infectious period differentiated between the high- and low-exposed individuals. Individuals with low infectiousness do not show symptoms and are difficult to identify, while those with high infectiousness do develop symptoms and are more easily detected. All other epidemiological parameters are assumed equal between both host types

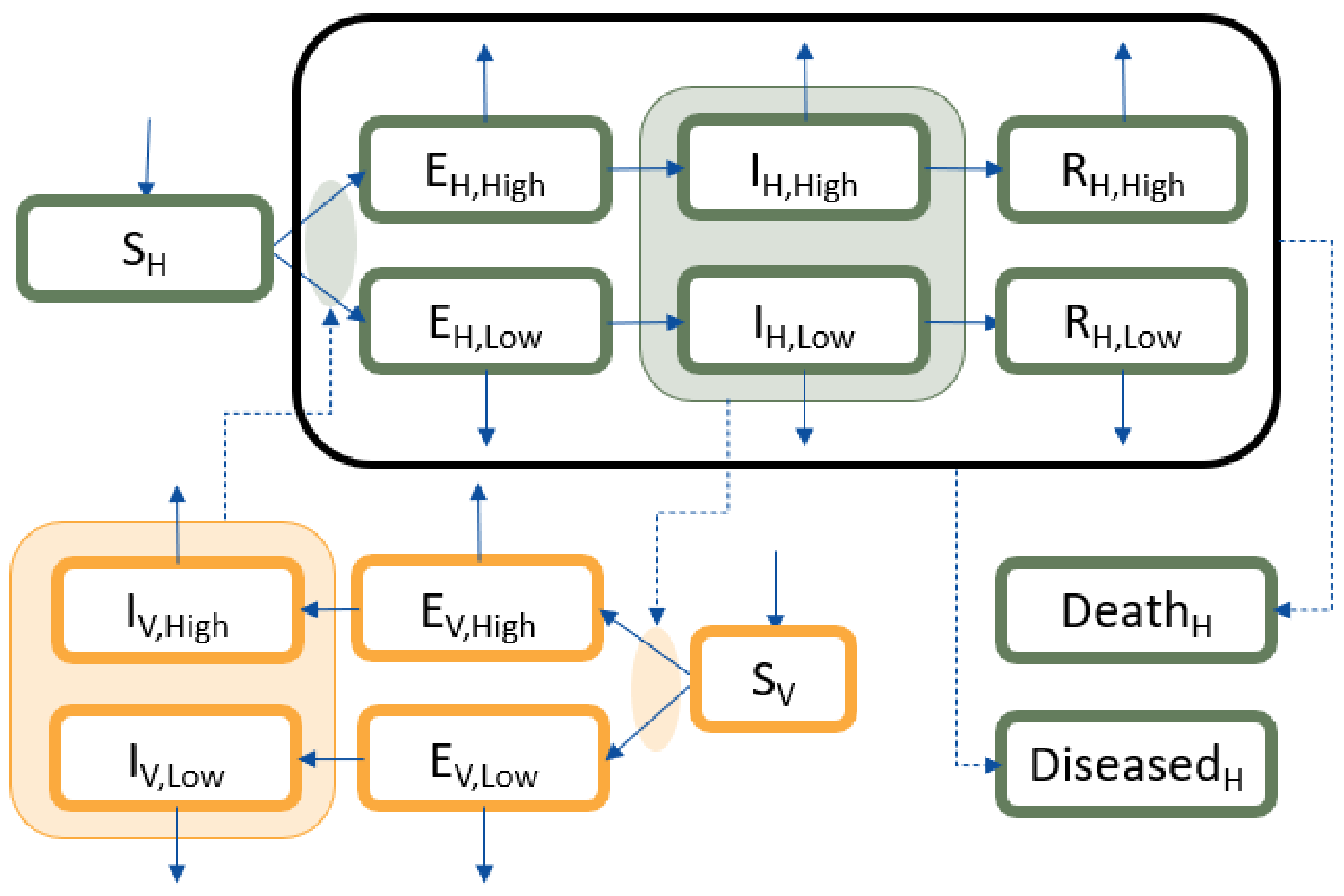

Figure 1. Compartmental model
Outline of the used model, S, E, I, and R denote the Susceptible, Exposed, Infectious and Recovered compartments. Subscripts H and V denote Host and Vector compartments.

Results

Figure 2 shows who will dominate the equilibrium reached. Based on the vector-to-host ratio and the relative infectiousness of high- and low-infectious hosts calculated as the number of vectors infected by one low-infectious host divided by the number of vectors infected by one high-infectious host on average. Figure A follows from introducing 1 high-infectious host in a disease-free system. Figure B shows the results for the situation where a low-infectious host was introduced into the system.

Figure 3 Shows, for different diseases systems, the the value of R0, as well as the percentage of infectious hosts per type in equilibrium and the chance of becoming low-infectious. On the left a disease where a high-infectious host infects on average 9.5 time as many vectors compared to a low-infectious host. On the right a system where a high-infectious hosts infects only 1.1 times as many vectors as a low-infectious host. The dotted lines show the forward plot, where one infectious vector is introduced in the disease-free equilibrium at varying vector-to-host ratios. The solid lines show the backward plot where vectors are sequentially removed from an endemic situation (starting at the right side of the plot).

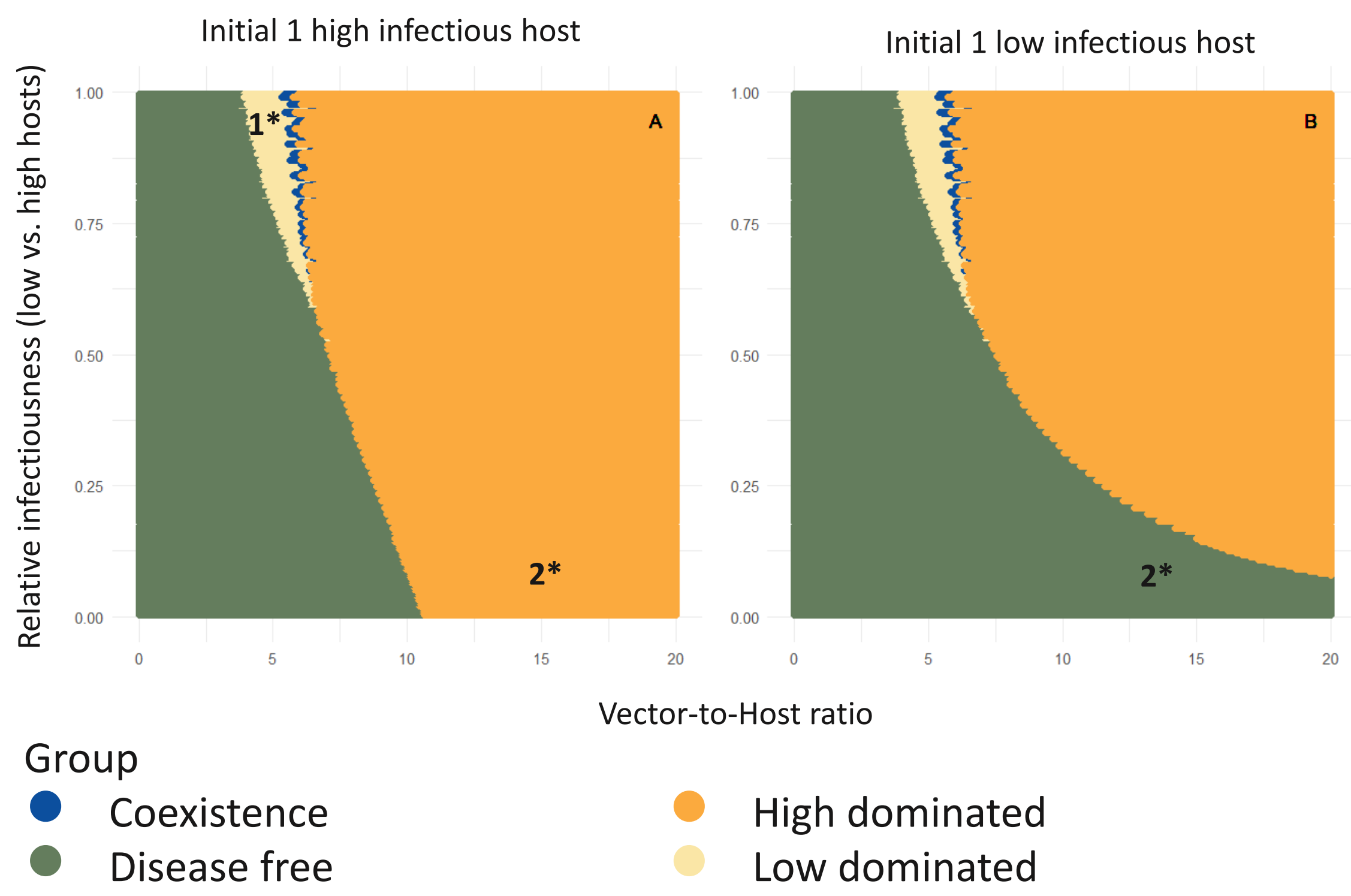

Figure 2: Infectious hosts in equilibrium

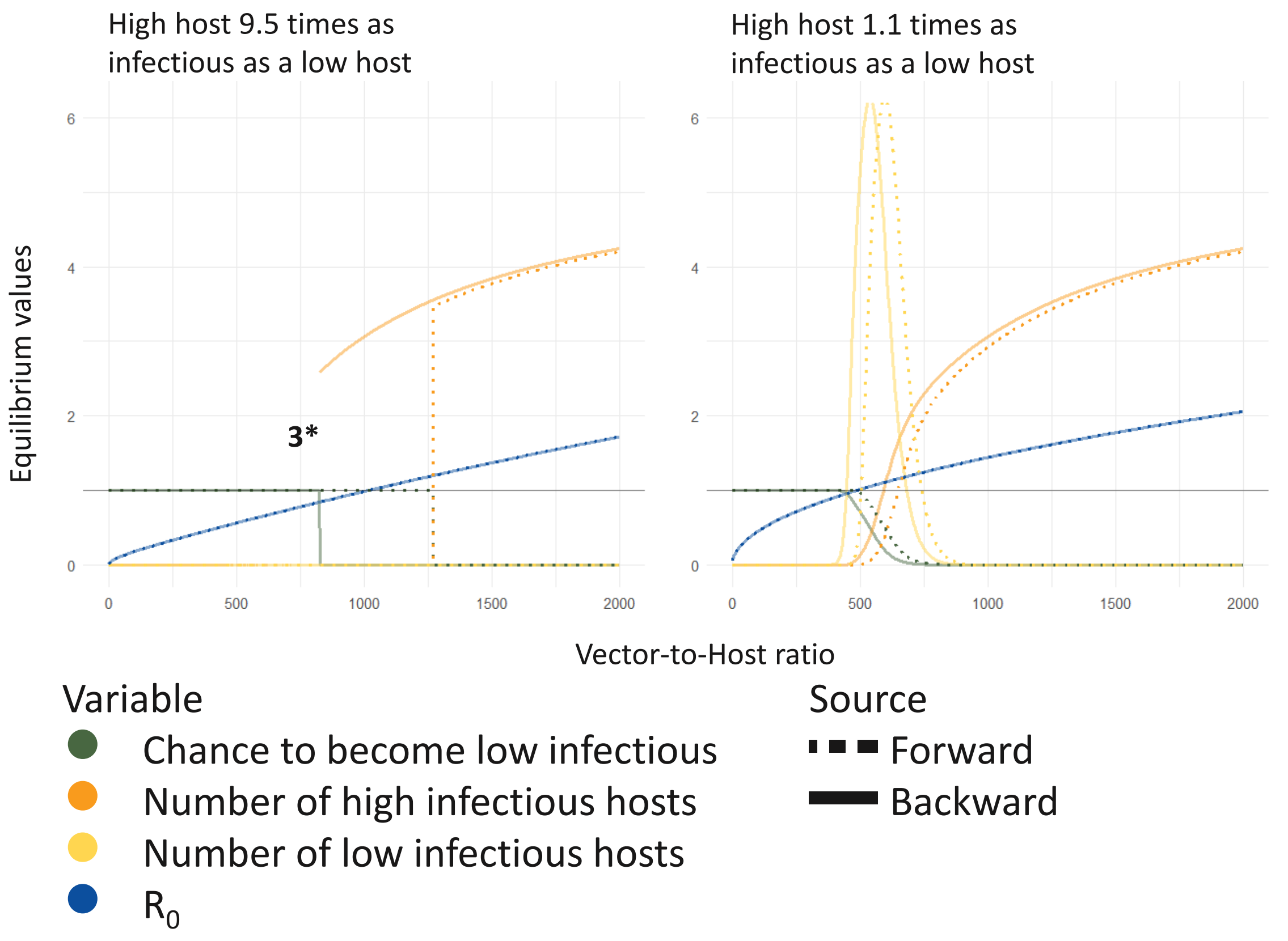

Figure 3: Bifurcation plots

Take Home

1. When the vector-to-host ratio is low, it is possible that low infectious animals dominate the equilibrium, causing silent transmission. (Figure 2, 1*)
2. The initial situation determines the equilibrium reached in systems where positive feedback is in place. (Figure 2, 2*)
3. Reducing the number of vectors to a level in which a disease cannot invade, does not mean this will eradicate the disease. (Figure 3, 3*)

Future direction: Validate the model using empirical data

References

1: Styer, L. M., Bernard, K. A., & Kramer, L. D. (2006). Enhanced early West Nile virus infection in young chickens infected by mosquito bite: effect of viral dose. *American Journal of Tropical Medicine and Hygiene*, *75*(2), 337-345.
2: Baylis, M., O'CONNELL, L., & Mellor, P. S. (2008). Rates of bluetongue virus transmission between Culicoides sonorensis and sheep. *Medical and veterinary entomology*, *22*(3), 228-237.

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