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## Introduction

Livestock disease can be tackled through a number of routes including vaccination, biosecurity, restricting animal movements to between accredited farms, and testing and quarantine during transit. Designing the most effective strategies benefits from accurate assessment of the herd-to-herd spread of disease.

However, the capacity for disease transmission within populations is frequently measured using the basic reproduction number,  $R_0$ , which provides a poor measure of disease spread in a highly structured population (e.g. a population of cattle herds), and says little about between-group transmission dynamics. Instead we examine the properties of  $R_{pop}$ , the expected number of secondary herds infected while the disease persists in the primary herd.

Previous studies featuring structured population household models have typically focused on *phenomenological* disease spread, which fails to account for depletion of infectives due to *mechanistic* disease transmission.

## Methods

We expect mechanistic movement to be important for the assessment of movement based control measures, and therefore build the model to address this.

Using a stochastic individual based metapopulation SIR model we measure  $R_{pop}$  and examine the effect of heterogeneity in herd sizes  $c$ , herd movement rates  $m$ , and individual infectiousness with mean  $\beta$  (e.g. different shedding rates and the 80-20 rule<sup>1:2</sup>), and examine the results of movement-based control options (e.g. testing and treating, or quarantining animals when they move between herds).

We solve the master equation to obtain the quasi-equilibrium distribution<sup>3</sup> of infectives when disease control coincides with cattle movement, and derive the following expression for  $R_{pop}$

$$R_{pop} = mNP_{pos}T_{inf}$$

where  $m$  is the movement rate,  $N$  is the herd size,  $P_{pos}$  is the mean prevalence in infected herds, and  $T_{inf}$  is the expected time until the infection dies out.

## Results

$R_{pop}$  peaks at intermediate movement rates.

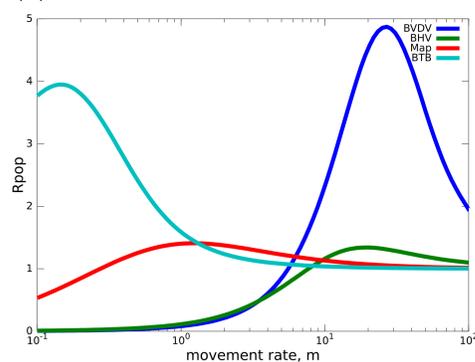


Figure 1:  $R_{pop}$  is shown for parameters representative<sup>4</sup> of BVDV, BHV, Map, and BTB. The peak of  $R_{pop}$  increases with disease transmission (horizontal and vertical), and herd size, and we expect movement based control to be most difficult at these peaks. Note that  $R_{pop} > 1$  is necessary for the disease to spread between herds and persist. For very high  $m$ ,  $R_{pop}$  tends to 1 in the absence of disease intervention, regardless of the other parameters.

Herd size heterogeneity increases  $R_{pop}$

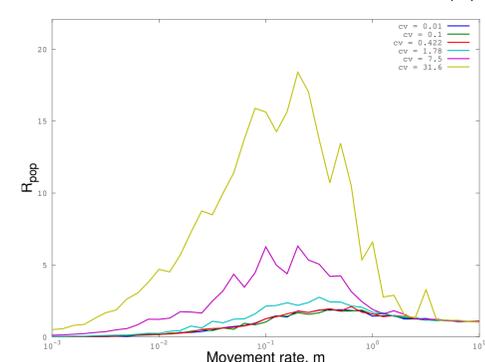


Figure 3: We examine heterogeneity in herd sizes by assigning herd sizes randomly distributed with mean  $c$  and dispersion index  $c_v$ , and varying the dispersion index.  $R_{pop}$  has nonlinear dependence on herd size, and is consequently increased by heterogeneity, with larger herds contributing more infectives, as disease in those herds has higher prevalence (larger  $N$  and  $P_{pos}$ ), and persists longer (larger  $T_{inf}$ ) due to lower chance of stochastic fadeout, and greater interaction with other herds. Disease control is therefore easier when herd sizes are more homogeneous.

Counter-intuitive responses to movement-based control measures

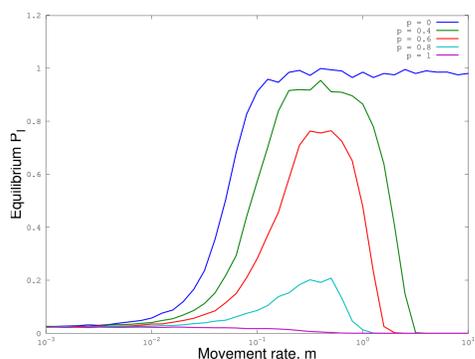


Figure 2: To examine response to movement based control, we introduce a parameter  $p$ , which gives the probability of an infective responding to disease intervention before arrival at a new herd. Disease intervention may only reduce  $R_{pop}$  to below 1 for certain ranges of movement rates, leaving islands where the disease persists. The interval where  $R_{pop} > 1$  differs for different disease parameters, so disease intervention on movement may be suitable for particular diseases, but not others, and that situation may be reversed if movement rates are adjusted.

Heterogeneity in infection and movement decreases  $R_{pop}$

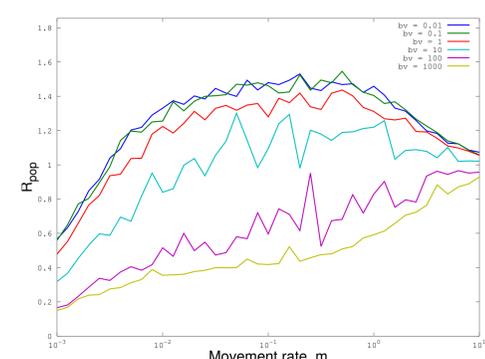


Figure 4: We examine heterogeneity in individual infectiousness by varying the dispersion index  $\beta_v$ .  $R_{pop}$  is reduced by heterogeneity in individual disease transmission, due to the greater chance of stochastic fadeout if the primary has low infectivity. Movement acts as transmission between groups, rather than individuals, and heterogeneity in herd movement rates typically reduces  $R_{pop}$ , however it can increase  $R_{pop}$  when  $m$  is very low.

## Discussion

Using structured metapopulation models, we see that  $R_{pop}$  peaks at intermediate movement rates. In combination with testing and treating prior to movement, or post movement quarantine, this can lead to islands of movement rates that permit the disease to persist. Under such control schemes, a reduction in movement rates can, counter-intuitively, lead to an increase in disease prevalence. Heterogeneity in herd sizes, movement rates, and individual infectiousness influences  $R_{pop}$ , which has important implications for disease control.

## Bibliography

- [1] Woolhouse, M. et al. 2005. Epidemiological implications of the contact network structure for cattle farms and the 20-80 rule. *Biology Letters* 1(3): 350–2.
- [2] Matthews L. et al. 2006. Heterogeneous shedding of Escherichia coli O157 in cattle and its implications for control. *Proc. Natl. Acad. Sci. USA*, 103(3): 547–52.
- [3] Nåsell, I. 1999. On the quasi-stationary distribution of the stochastic logistic epidemic. *Mathematical Biosciences*. 156: 21–40.
- [4] Carlslake D. 2011. Endemic cattle diseases: comparative epidemiology and governance. *Phil. Trans. R. Soc. B*. 366: 1975–86.