# How does increasing immunity change spread kernel parameters in subsequent outbreaks? – A simulation study on Bluetongue Virus

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# How does increasing immunity change spread kernel parameters in subsequent outbreaks? – A simulation study on Bluetongue Virus.

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Modelling the spatial spread of vector borne diseases, one may choose methods ranging from statistic to process oriented. One often used statistic tool is the empirical spread kernel. An empiric spread kernel fitted to outbreak data provides hints on the spread mechanisms, and may provide a good estimate on how future epidemics could proceed under similar conditions. However, a number of variables influence the spread of vector borne diseases. If one of these changes significantly after an outbreak, it needs to be incorporated into the model to improve the prediction on future outbreaks. Examples of such changes are: vaccinations, acquired immunity, vector density and control, meteorological variations, wind pattern, and so on. Including more and more variables leads to a more process oriented model. A full process oriented approach simulates the movement of virus between vectors and host, describing density and motion of vectors/hosts, climatic variables, and so on will theoretically be able to describe an outbreak under any circumstances. It will most likely contain parameters not very well established, and is also very heavy in computer time. Nevertheless, we have tried to create a relatively detailed simulation spread model. And by using empirical spread kernels from past outbreaks we have fitted some of the more uncertain parameters for this case study.

A stochastic simulation model was developed for the spread of bluetongue virus. In the model hosts (cattle) and vectors (*Culicoides*) are distributed onto a grid representing farm/field quadrants of 1 hectare. Each quadrant has a host SEIR model (Susceptible Exposed Infectious Recovered) and a vector SEI model attached. Transmission of virus between hosts and vectors depend on many parameters most of which are temperature dependent. Spatial movement of virus between quadrants is modelled by local flight and wind spread of vectors.

The simulated spatial spread rate of virus is very dependent on movement parameters, but also the distribution and total numbers of hosts and vectors influenced the spread of virus. With empirical spread kernels from past outbreaks and known distributions of host animals, it was possible to fit parameter values of vector movement.

The final model including the fitted process based movement parameters is used to simulate e.g. 50% of cattle protected by acquired immunity after a first epidemic outbreak. We can then demonstrate how this changes the spread kernel for future outbreaks.