

# Analysis of FMD control strategies using contact network simulations



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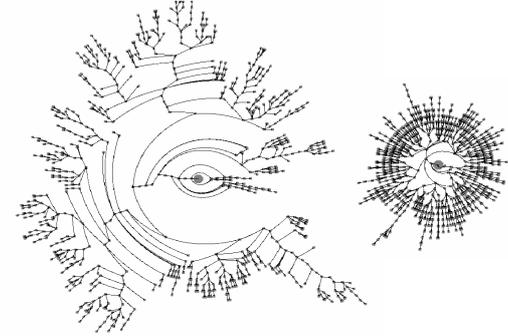
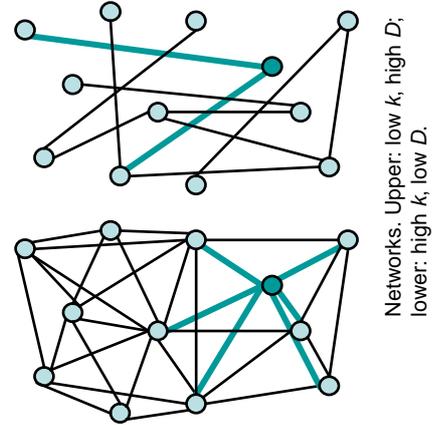
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After the UK 2001 FMD epidemic, there was much discussion over the relative benefits of disease control strategies, particularly of the dangerous contact (DC) and contiguous premises (CP) culls. Which is better depends upon both the ability to identify DCs, and the number of potential DCs per infected premises (IP).

One DC that always results in transmission is very different from ten that transmit only 10 % of the time. In the first case, identifying a DC eliminates all transmission; in the second case only 10 % of it. CP culling relies on link length: the more clustered links are, the more valuable CP culling becomes.

We represent contact using networks [right], with numbers of potential infectious links “ $k$ ” (i.e. potential DCs) and clustering parameter “ $D$ ”. We examine the UK 2001 data to determine ranges of  $k$  and  $D$  consistent with the epidemic, and relate this to the efficacy of culling on farming networks.

First, we consider the “forward” problem: to what extent does model output vary across networks with different underlying contact structures?

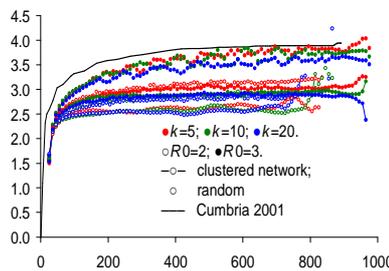


Epidemic trees from simulated epidemics.

Left: clustered network: right, random network.

A comparison of network-based models (Green et al., 2005, submitted) showed that, while different architectures can produce epidemics with the same overall structure, the underlying models of transmission required to do this are different:

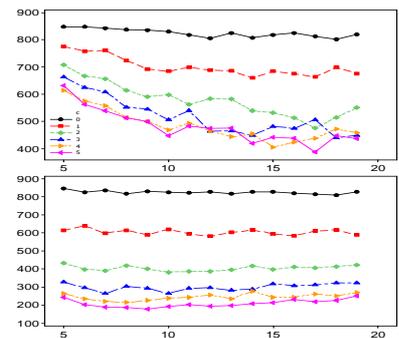
There are relationships amongst changes in the infectiousness of IPs over time, the value of  $k$ , and the distribution of generation times.



Tree index B2 versus epidemic size for simulated epidemic data and that from UK 2001 Cumbria.

IPs and their source infections were represented by epidemic trees of descent [far left]. Trees showed that a range of model architectures fit a single epidemic.

Indices of tree shape for Cumbria 2001 were consistent with unclustered networks and an  $R_0$  of 3 [left].

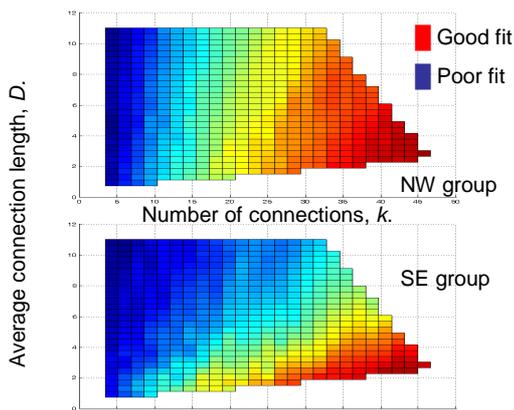


Total removals (out of 1000) v.  $k$  for different culling levels  $c$ . Upper: CP; Lower: DC cull. Clustered networks.

Though exact determination of  $k$  might be impractical, hypothetical scenarios can be modelled, and the impact of different disease control strategies examined. Both DC and CP culling were implemented in the model.

Differences in the efficacy of culling were found with different values of  $k$  – even in cases where with no control, model output was insensitive to  $k$  (Green et al., 2005). For CP culling on clustered networks, number of culled premises was negatively correlated with  $k$  [cf. DC culling, right]. For DC culling on random networks, the correlation was positive.

Culling must be prompt, both for IPs and especially so for DC culling on random networks, where a delay of one day results in 30 % more removals for small  $k$ .



How possible is a given epidemic for a particular network structure? Here, we consider Cumbria in 2001, subdivided into two sections: an earlier series NW of Penrith, and a later series SE [left].

Differences were found concerning clustering. In the SE group, more clustered networks (low  $D$ ) explain the epidemic better, suggesting short-range transmission. In the NW group, clustered and unclustered networks fitted equally well – either epidemic spread was truly random; or because of the high density of IPs, spreading patterns cannot be discerned.

**To conclude:** The contact network of an epidemic is important in the comparison of different control policies. The inability to distinguish between models with different contact structures suggests that they alone cannot be used to distinguish between CP and DC culls, unless more sophisticated analysis techniques are used.

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