

# Electronic Supplementary Material for Modelling the Propagation of Social Response during a Disease Outbreak

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## 1 Network structure

For model simulations, the disease spread graph was a Watts-Strogatz random graph [1]. This random graph is commonly referred to as a small-world network, because it has strong local clustering but short average path lengths, resulting from a small number of random contacts. For our purposes, the local structure of the graph represented regular contacts, such as those with classmates, colleagues and family members. The random contacts represented transmission of disease between strangers. Like the disease, social response travels through connections between friends and colleagues. However, unlike disease, social response communication is not restricted to interactions in which both parties are physically present. Non-physical interactions through social media, email or telephone were represented in the model by a random scale-free graph. This type of graph has a degree distribution that follows a power law [2]. A few high degree nodes are connected to much of the population, while most nodes have only a few connections. It is believed that the world wide web and collaboration networks resemble a scale-free graph [2]. In summary, the social graph was a scale-free graph overlaid on top of the small-world disease graph and included all edges from the disease spread graph, as well as additional edges representing remote interactions. All agents in the social graph were given a self-loop, that is, for each agent  $i$ ,  $(i, i)$  was an edge in the social graph. The self-loop ensured that agents always included their own opinions in the social response update.

## 2 Model fitting and parameter selection

We fit the parameters for the case study simulations by first making observations about disease severity and incidence and then selecting parameters that provided a good fit to the observed data.

### 2.1 Case study: Hong Kong SARS and H1N1

For modelling H1N1 in Hong Kong, the perceived disease risk was low ( $\kappa = 0.60$ ), while the incidence was relatively high. We estimated  $\kappa$  through manual analysis of multiple data sources and used the same value for the Hong Kong H1N1 and fall Mexico City outbreaks as they occurred concurrently and H1N1 was already a familiar disease. SARS was novel and severe ( $\kappa = 0.95$ ), but with low incidence. For fitting the SARS disease spread, we selected disease parameters ( $\beta$ ,  $T_R$ ,  $\eta$  and  $\tau$ ) such that the simulated expected number of new infections would approximately match the number of confirmed new cases, rescaled to a population of 400,000. For H1N1, we selected disease parameters such that 11% of the population of 400,000 agents would become infected. After setting the disease spread parameters, we modelled the projected social response for both cases.

## 2.2 Case study: H1N1 in Mexico City

We modelled the distinct dynamics of the spring and fall outbreaks of H1N1 in Mexico City by selecting the disease spread parameters, ( $\beta$ ,  $T_R$ ,  $\eta$  and  $\tau$ ) and the disease risk index ( $\kappa$ ). The disease spread parameters were set such that the expected new infections matched the confirmed new infections per day. The size of disease was scaled, so that the 400,000 agent network could stand in for Mexico City, which has a population of about 9 million. Additionally, since confirmed cases represented approximately 3.5% of total cases in Hong Kong, we assumed that 3.5% of total cases were confirmed in Mexico City. For the spring wave of H1N1 infection, we set  $\kappa = 0.75$  to reflect the novelty of H1N1, its appearance outside of the normal flu season, its perceived severity in light of the La Gloria outbreak and the fact that severe cases had been seen in young people, which is indicative of a more virulent form of influenza. For the fall wave of H1N1 infection, the disease risk index was reduced to  $\kappa = 0.60$ , since the population had become more familiar with H1N1 and because it had been shown that H1N1 was not as severe as originally feared. The media penetration, communication and decay parameters ( $p$ ,  $q$  and  $\alpha$ ) were not varied between simulations.

## References

- [1] Watts D, Strogatz S. Collective dynamics of ‘small-world’ networks. *Nature*. 1998 Jun 4; 393(6684): 440-442. (DOI: 10.1038/30918.)
- [2] Newman MEJ. *Networks: An introduction*. Oxford; 2010.