A population model of interacting SARS-CoV-2 variants

Peter Mann,* V. Anne Smith, John B.O. Mitchell, and Simon Dobson
School of Computer Science, University of St Andrews, St Andrews, Fife KY16 9SX, United Kingdom
EaStCHEM School of Chemistry, University of St Andrews,
St Andrews, Fife KY16 9ST, United Kingdom and
School of Biology, University of St Andrews, St Andrews, Fife KY16 9TH, United Kingdom
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I. ABSTRACT

We have developed an analytical model that can be used to understand the role that contact structure and inhomogeneous susceptibility (caused by vaccination or demographics) have on the spread of emergent, temporally separated variants of SARS-CoV-2 using an N-variant network model [1–3].

At the time of writing, SARS-CoV-2 has proven to evolve on a timescale relevant for vaccine development and distribution. It is unknown precisely how the dynamics of an emergent phenotype will unfold over a given, possibly dynamic, population structure. Further, it is unknown how the novel variant will interact with individuals, or groups that have inhomogeneous susceptibility either through vaccination, previous exposure to earlier variants of SARS-CoV-2 or demographics such as age, gender or underlying health conditions. We can gain insight into how these diverse factors affect the dynamics of each variant by using a population model based on a complex network. A network model provides an excellent opportunity to blend uncertainties arising from the contact structure and the nature of the interaction of a given variant with previous variants or vaccination levels.

In our work we have introduced an analytical framework for an N-variant population model of disease spreading based on sequential Susceptible-Infectious-Removed (SIR) processes. As each process spreads across the population, individuals are either infected or uninfected by that variant; giving each person an *infection history*. When subsequent variants spread over the population, the infection history of each person influences how likely it is that the latest variant will infect them. The nature of the influence can be entirely tailored to each unique history via a smoothly varying parameter; ranging from antagonistic cross-immunity to facilitatory coinfection. Our model can therefore be used to understand the role that contact structure and disease interaction play in shaping the dynamics of an emergent variants of the SARS-CoV-2 pathogen.

The structure of the contact network is a parameter of the model. For example, we can consider random graphs whose degrees are Poisson distributed or those that exhibit power-law scaling that have high-degree hubs. We can insert particular subgraphs, such as cycles or cliques; the structure of which might be mined from empirical networks. Similarly, we could model the impact of mitigation strategies, such as social distancing or mask wearing, by creating random graphs with an appropriate topology, perhaps multi-layered or modular.

An assumption of the model is that the variants are temporally separated; indicating that the dynamics of one process has finished before the next variant emerges. Therefore, disease interaction is unidirectional: later variants do not influence the spread of previous ones.

The model is constructed using the combinatorial exact method of generating functions [4]. The generating function technique represents all possible outcomes of a disease, weighted by the probability of their occurrence. From this, ensemble averages that represent global dynamics can be constructed.

* pm78@st-andrews.ac.uk

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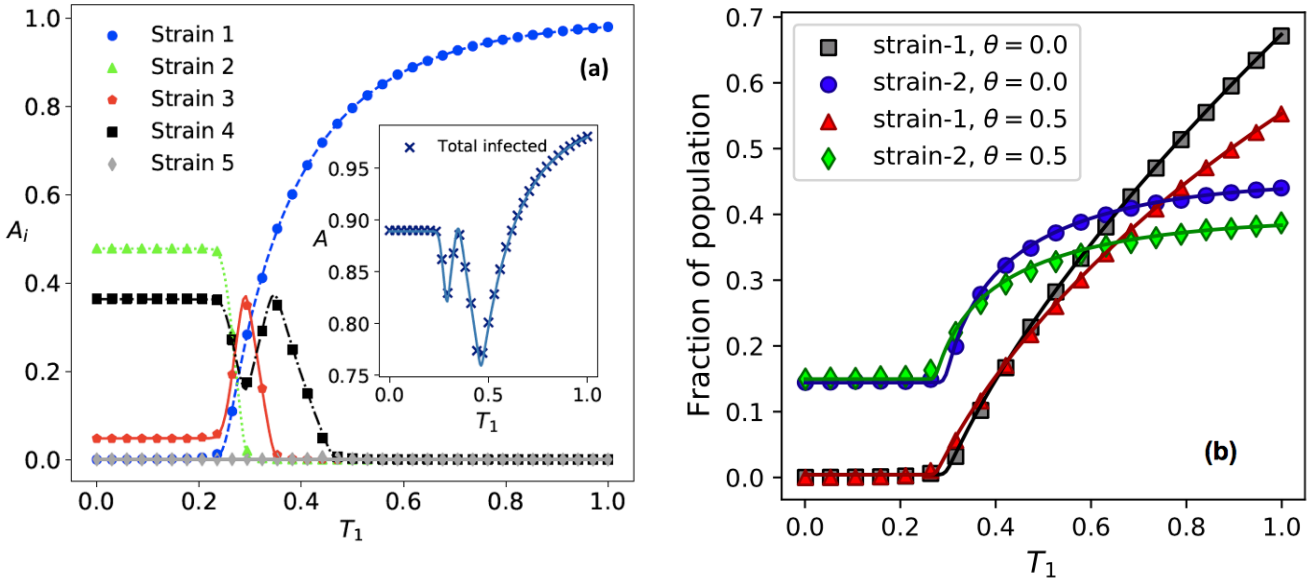

FIG. 1: (a) The outbreak sizes $\mathcal{A}_i$ of subsequent variants as a function of the transmissibility of the first variant, $T_1 \in [0, 1]$. Each pathogen has a strongly antagonistic interaction with previously infected individuals such that prior infection by a previous variant prevents future infection. The inset shows the that the total number of infected people is not a constant. In this example the parameters are $T_2 = 0.35$, $T_3 = 0.5$, $T_4 = 1.0$ and $T_5 = 1.0$ for a Erdős-Renyi random graph with mean degree $\langle k \rangle = 4$ and $N = 30000$ nodes. Scatter points are the average of experimental stochastic simulations whilst the plotted lines are the results of the model. (b) The outbreak fractions for two variants for clustered and unclustered networks as T_1 is varied over the unit interval with a partial coinfection disease coupling $(T, T') = (0.4, 0.7)$. Clustering is controlled by the probability θ that a triangle is formed from a triple of edges.