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Pairwise approximations of simplicial contagion models on hypergraphs

Keywords: epidemiology, dynamical networks, higher-order interactions, complex networks, disease spread

Extended Abstract

Since the first theoretical compartmental models of disease spread proposed by Kermack & McKendrick, the study of contagion models have consistently increased in research interest. These aforementioned ODE models are limited to the assumption that populations homogeneously mix. However, rarely is this the case in real world occurrences of contagion where spreading dynamics interact heterogeneously on some complex topology such as a social contact network. Crucially, local properties and structure can mediate non-trivial dynamics as observed in historical epidemic outbreaks such as avian influenza [1] and COVID-19 [2].

Fortunately, spreading mechanisms of classical compartmental models can be extended to complex networks where individuals – represented as nodes – transition between disease states depending on the states of their direct neighbours [3]. This generalisation aims to provide more realism by allowing a contact process to guide disease spreading dynamics. However, this assumes that agents are restricted to pairwise interactions and do not account for group interactions or the effect of local infection concentration on spreading dynamics, both plausible when one considers individual tendencies to interact in social groups.

In an effort to explore more complex and realistic dynamics, recent simplicial contagion models (SCM) propose the inclusion of network interactions along higher-order topologies such as simplices (e.g. triangles, tetrahedrons) to account for social interactions that include more individuals than pairwise interactions. The inclusion of these higher-order interactions have been shown to promote critical transitions and bistability in epidemics [4]. However, the importance and uniqueness that these higher-order topologies play in altering the overall epidemic trajectory is less clear.

We present a numerical agent-based model (ABM) that unifies the classical ODE compartmental model and SCM approaches and extends it to interactions on hypergraphs with simplices of arbitrary order. Using this model, we investigate to what extent higher-order interactions govern observed disease dynamics.

We find that network topology primarily affects transient epidemic dynamics. If the network structure is fully known, one can calculate normalisation values related to the potential network activity. These values may be used to define disease parameters for first-order pairwise interactions that replicate the steady state dynamics of those that include higher-order interactions (see Figure 1). Additionally, we find a duality where the effects of network topology can be overcome by allowing for temporally varying disease parameters; allowing first-order pairwise interactions to closely approximate both transient and steady state trajectories of higher-order systems (see Figure 2).

The ability for pairwise interaction models to reproduce epidemic trajectories from models containing higher-order complexity features raises interesting questions on the need for higher-order network topologies, which may arise from various socio-cultural interaction patterns, to be included in disease models. Furthermore, it encourages a reconsideration of whether dynamics, observed or modelled, should be attributed as the result of complex topological structure or temporally varying spreading mechanisms.

References

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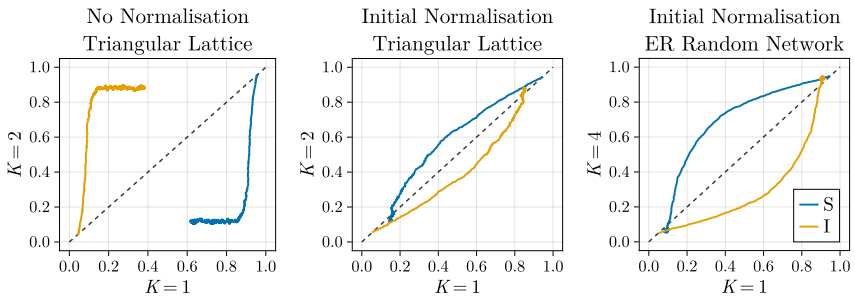

Figure 1: Comparison of susceptible-infected (SI) epidemic trajectories between the pairwise and higher-order simulations. Unnormalised case where the base infection rate β of both simulations are equal (left), spread on a triangular lattice (middle) and random network (right) where infection rate of the pairwise case is increased such that network activities between the lower and higher-order case are approximately equal. Normalisation allows pairwise simulations to replicate the steady-state behaviour of simulations with higher-order interactions.

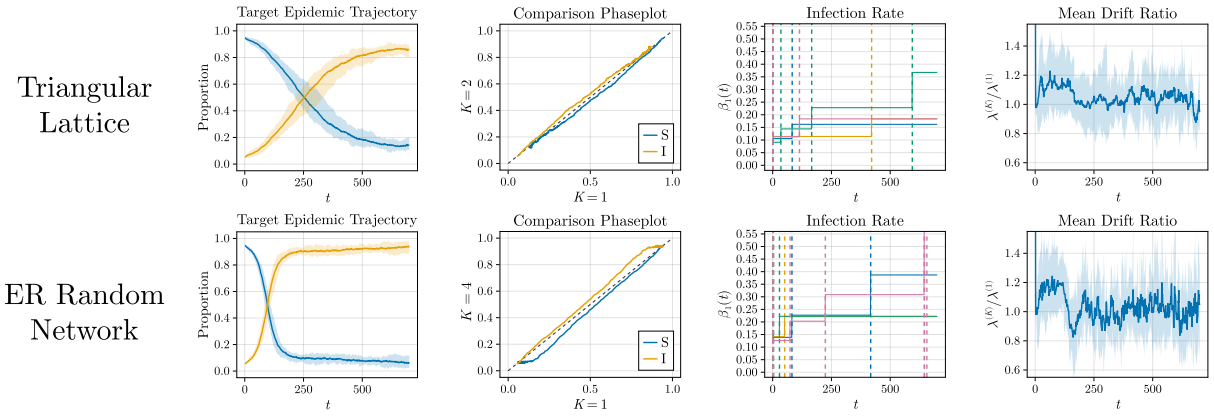

Figure 2: Simulations using dynamically adjusted disease parameters to allow pairwise interactions to replicate higher-order dynamics for a triangular lattice and Erdös-Rényi (ER) random network. K is the maximal clique simplex order included in the simulation. Shaded regions are 90% CIs. From left to right: target trajectories from the higher order simulation, comparison trajectories between the pairwise case $K = 1$ and higher-order case, randomly chosen trajectories of the dynamic infection rate $\beta_1(t)$ with adjustment events indicated by vertical lines, and the ratio of the mean network activity of between the high and lower order scenarios $\bar{\xi}(t)$.