Spatial effects on epidemics diffusion: Network topological characteristics leading to power-law time-dependent growth

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During the initial phase of the covid-19 epidemic, it has been observed that the pathogen diffusion is characterized by a power-law growth [1, 2]. This sub-exponential increase appears independent of geographical or socio-economic parameters, given that it is shown for several China provinces and European countries. Historically, a quadratic diffusion has been also reported for other epidemics, such as AIDS. The persistence of such scaling in diverse systems sets a challenge for epidemiological modeling. In the current work, we first note that power-law scaling is a general diffusion characteristic not restricted to epidemics among humans. In particular, we juxtapose the Covid-19 and the AIDS epidemics to highly contagious disease in animal livestock, by revisiting foot-and-mouth data from the 1967 and 2001 outbreaks in the UK. We find similar power exponents over the initial diffusion period, even before mitigation measures been taken to reduce contagion. Traditionally, efforts to address diffusion characteristics like the sub-exponential growth or a reduced population size in herd immunity are considered in the framework of "fully mixed" models (such as SIR or SEIR). The aim is to reduce the presumed efficiency of the susceptible "S" or infected "I" populations in the contagion process in a variety of ways, such as: By introducing nonlinearity in the S and/or I interaction terms of the model [3]; by assuming heterogeneity in the susceptibility or the transmissivity of the populations [4]; by inferring a particular generic transmission mechanism [1, 5]. In our approach, however, in the framework of "agent based models", we note that such assumptions may not be needed: A power-law scaling may well be a purely geometric effect, resulting from spatial/topological characteristics of the associations among the involved agents [6]. We demonstrate this in numerical simulations on 2-D Cartesian lattices, with and without small world structure, where contagion is spread in various ways: When infection progresses from nearest and next nearest neighbors; from long-distant neighbors; due to external influence that infects random sites. In all cases studied, we find a Weibull closed form for the aggregated disease propagation, which is reduced to a power-law growth at small times. Using this analytic tool we also discuss the spatial disease spread in interconnected geographical regions, where a Pareto distribution has been observed [7]. We thus argue that spatial/topological characteristics of the associations among agents involved in the contagion should be explicitly taken into account when analyzing basic features of the epidemics diffusion.

References

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