Abrupt phase transition of epidemic spreading in simplicial complexes

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Recent studies on network geometry, a way of describing network structures as geometrical objects, are revolutionizing our way to understand dynamical processes on networked systems. Here, we cope with the problem of epidemic spreading, using the susceptible-infected-susceptible (SIS) model, in simplicial complexes. In particular, we analyze the dynamics of the SIS in complex networks characterized by pairwise interactions (links) and three-body interactions (filled triangles, also known as 2-simplices). This higher-order description of the epidemic spreading is analytically formulated using a microscopic Markov chain approximation. The analysis of the fixed point solutions of the model reveals an interesting phase transition that becomes abrupt with the infectivity parameter of the 2-simplices. Our results pave the way to advance in our physical understanding of epidemic spreading in real scenarios where diseases are transmitted among groups as well as among pairs and to better understand the behavior of dynamical processes in simplicial complexes.

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The collective behavior of dynamical systems on networks, has been a major subject of research in the physics community during the last decades [1–5]. In particular, our understanding of both natural and man-made systems has significantly improved by studying how network structures and dynamical processes combined shape the overall system behavior. Recently, the network science community has turned its attention to network geometry [6–9] to better represent the kinds of interactions that one can find beyond typical pairwise interactions.

These higher-order interactions are encoded in geometrical structures that describe the different kinds of simplex structure present in the network: a filled clique of \( m \) nodes is known as an \( m \)-simplex, and together a set of 1-simplices (links), 2-simplices (filled triangles), etc., comprise the simplicial complex. While simplicial complexes have been proven to be very useful for the analysis and computation in high dimensional data sets, e.g., using persistent homologies [10–14], little is understood about their role in shaping dynamical processes, save for a handful of examples [15–18].

A more accurate description of dynamical processes on complex systems necessarily requires a new paradigm where the network structure representation helps to include higher-order interactions [19]. Simplicial geometry of complex networks is a natural way to extend many-body interactions in complex systems. The standard approach so far consists in understanding the coexistence of two-body (link) interactions and three-body interactions (filled triangles). Note that this approach is different from considering pairwise interactions among three elements of a triangle, it refers to the interaction of the three elements, in the filled triangle, at unison.

Here we present a probabilistic formalization of the higher-order interactions of an epidemic process, represented by the well-known susceptible-infected-susceptible (SIS) model [20] in one- and two-simplices, revealing that the consideration of higher-order structure (filled triangles) can change the character of the phase transition of the epidemic spreading to the endemic state. Specifically, we find that for a significant region of the parameters space the continuous phase transition that has been well-characterized in networks so far [5] becomes abruptly discontinuous [21]. These results are important for physicists working on network science, independently on the particular dynamical process we have chosen, to explain why this physical phenomena could arise in complex systems. Similar results have been reported for social contagion dynamics [17] using a mean-field approach.

For the mathematical formalization of the dynamical process, we use simplicial complexes extensions of the microscopic Markov chain approach (MMCA) [22–27], and of the epidemic link equations (ELE) [28], that compute the probabilities of 1 and 2-simplices to transmit the epidemics. This formalism allows us to get physical insight into the phase transition and its consequences at the level of our understanding of plausible epidemic scenarios.

Let us start by considering the dynamics of the SIS model in networks. We consider a network of \( N \) nodes and \( L \) links, where the nodes can have two states, susceptible (S) or infected (I). The classical interaction so far considers that the infection propagates, pairwise, from certain infected individuals to their neighbors with a probability \( \beta \), and infected nodes eventually recover with probability \( \mu \). In the simplicial complex scenario, we will consider also triangular interactions, i.e., every node also interacts within the 2-simplices with the two neighbors at unison, with an infection probability \( \beta^\Delta \) when the other two members are infected. We can define a system of \( N \) discrete-time MMCA equations that capture the...
evolution over time of the probability $p_i$ of node $i$ being in an infected state as
\begin{equation}
p_i(t+1) = (1-p_i(t))(1 - q_i(t)q_i^\triangle(t)) + p_i(t)(1-\mu),
\end{equation}
where $q_i(t)$ defines the probability that node $i$ is not infected by any pairwise interaction with its neighbors,
\begin{equation}
q_i(t) = \prod_{j \in \Gamma_i} (1 - \beta p_i(t)),
\end{equation}
and $q_i^\triangle(t)$ is the probability that node $i$ is not infected by any of its interactions at the 2-simplicial level,
\begin{equation}
q_i^\triangle(t) = \prod_{j,k \in \Delta_i} (1 - \beta^\triangle p_j(t)p_k(t)).
\end{equation}
Here, $\Gamma_i$ and $\Delta_i$ represent the sets of 1 and 2-simplexes containing node $i$, respectively.

The system of equations (1) updates the probability of a node $i$ being infected as the probability of being susceptible at time $t$ and becoming infected by some neighbor, or some group of neighbors in a triangle [first term on the right-hand side (r.h.s.) of the equation], or the probability that node $i$ was already infected at time $t$ and it does not recover. This system of equations is a contraction map $T_{\beta,\beta^\triangle,\mu} : \vec{p}(t) \rightarrow \vec{p}(t+1)$ for every value of the parameters, and then the existence of fixed points is guaranteed by the Banach fixed point theorem [29]. We can solve the system by iteration. A naïve approximation of Eq. (1) is the homogeneous assumption in which all nodes have the same degree ($k$), belong to the same number of 2-simplexes ($k^\triangle$), and have the same probability of being infected ($p_i(t) = \bar{p}(t)$). Expanding up to second order in $p$, and developing the equation at the stationary state ($p(t) = \bar{p}$), it reveals the structure responsible for the abrupt transitions we could foresee:
\begin{equation}
(1-p)[\beta kp + (\beta^\triangle k^\triangle - \beta^2 k(k-1)/2)p^2] - \mu p = 0.
\end{equation}

This third-order algebraic equation has a trivial solution, $p = 0$, and two more roots, that depending on the parameters, provides up to two additional physical solutions with $p \in (0,1]$. For the parameters with three physical solutions, the stability analysis shows that the middle one is unstable, thus being responsible of the abrupt transitions, see Fig. 1. This result is equivalent to that obtained in [17].

Note, however, that Eq. (1) and in turn Eq. (4) carries the implicit assumption that the probability of being infected by one neighbor is independent on the probability of being infected by any other neighbor. This assumption is a mean-field approximation, whose validity is severely compromised in the current scenario, where we account for the infectivity in triads, and hence neighbors are unlikely to be independent.

To palliate the previous limitation, we can define the simplicial epidemic model at a level of links using a system of $3L$ equations. The size of this system is very large compared to the previous MMCA system of $N$ equations at the level of nodes; however, we can simplify the model to a system of $L+N$ equations if we consider the constrains imposed by the probabilistic model, i.e., the node marginal probabilities have to be the same regardless the link that we consider to compute them. These restrictions read as follows: for a link connecting nodes $i$ and $j$, the probability of a node $i$ to be in the susceptible state is $P_{iS} = P_{ijS}^S + P_{ijS}^L$, where $P_{ijS}^S$ is the joint probability of node $i$ being susceptible and node $j$ being susceptible, and $P_{ijS}^L$ is the joint probability of node $i$ being susceptible and node $j$ being infected. Equivalently $P_i^S = P_{iij}^S + P_{ij}^L$. Wrapping up these restrictions we can write the epidemic link equations, ELE [28], for the simplicial model, for every node $i$ as
\begin{equation}
P_i^S(t+1) = (1-P_i^S(t))(1-q_i(t)q_i^\triangle(t)) + P_i^S(t)(1-\mu),
\end{equation}
where $q_i(t)$ defines the probability that node $i$ is not infected by any pairwise interaction with its neighbors,
\begin{equation}
q_i(t) = \prod_{j \in \Gamma_i} \left(1 - \frac{P_{ijS}^S(t)}{P_{ij}^S(t)}\right),
\end{equation}
and $q_i^\triangle(t)$ is the probability that node $i$ is not infected by any of the interactions at the 2-simplicial level,
\begin{equation}
q_i^\triangle(t) = \prod_{j,k \in \Delta_i} \left(1 - \frac{P_{ijkS}^S(t)}{P_{ij}^S(t)}\right).
\end{equation}
Note that, to write down the previous equations, we have made use of Bayes’ theorem, substituting the conditional
probabilities $p_{j|i}^{PS}$ of node $j$ to be infected knowing that node $i$ is susceptible, by the joint probability $p_{i|j}^{PS}$; equivalently, for the 2-simplicial terms, $p_{j|i}^{PS} = p_{ijr}^{PS}/p_{i}^{S}$.

The system still requires of $L$ equations, one for every link, that account for the probability of having a link connecting two nodes in the infected state $Ii$, transitioning from the four possible states $SS$, $SI$, $IS$, $II$. It reads

$$
P_{jj}(t + 1) = p_{ij}^{SS}(t)(1 - q_{ji}(t)q_{ji}(t))(1 - q_{ji}(t)q_{ji}(t))$$
$$+ p_{ij}^{SI}(t)(1 - (1 - \beta)q_{ji}(t)u_{ji}(t)q_{ji}(t))(1 - \mu)$$
$$+ p_{ij}^{IS}(t)(1 - (1 - \beta)q_{ji}(t)u_{ji}(t)q_{ji}(t))(1 - \mu)$$
$$+ p_{ij}^{II}(t)(1 - \mu)^2, \quad (8)$$

where we have used: the probability of node $i$ not being infected by any neighbor different from $j$ through a link,

$$q_{ij}(t) = \prod_{r \in \Delta_i \setminus j} \left(1 - \beta \frac{p_{ijr}^{PS}(t)}{p_i^{S}(t)}\right); \quad (9)$$

the probability of node $i$ not being infected by any 2-simplex not containing node $j$,

$$q_{ji}(t) = \prod_{r \in \Delta_j \setminus i} \left(1 - \beta \frac{p_{ijr}^{PS}(t)}{p_i^{S}(t)}\right); \quad (10)$$

and the probability of node $i$ not being infected by any 2-simplex containing node $j$,

$$u_{ij}(t) = \prod_{r \in \Delta_i \setminus j} \left(1 - \beta \frac{p_{ijr}^{PS}(t)}{p_i^{S}(t)}\right). \quad (11)$$

See an illustration of the different contributions to Eqs. (9) to (11) in Fig. 2. To solve the system of Eqs. (5) and (8), we still need a closure for the ternary joint probabilities $p_{ijr}^{PS}$ found in Eqs. (10) and (11). To break the hierarchy of clusters produced by these terms we must rely on approximations. The classical pair approximation in statistical physics [30], also used in the context of epidemics by Mata and Ferreira [31], would consist in approximating $p_{ijr}^{PS} \approx p_{ij}^{PS}p_{ijr}^{PL}/p_{i}^{L}$. However, Cator and Van Mieghem [32], propose a different closure $p_{ijr}^{PS} \approx p_{ij}^{PS}p_{ijr}^{PL}$. The main problem with these proposal in the current scenario is that the symmetry of the elements in the 2-simplexes is broken, and this occurs in a degeneration of solutions. For example, using the second option, we could choose between three different closures for $p_{ij}^{PS}, p_{ij}^{PS}p_{ij}^{PL}, p_{ij}^{PL}, p_{ij}^{PS}p_{ij}^{PL}$, or $p_{ij}^{PL}$. Each alternative makes use of one node and one link (the link opposite to the chosen node in the triangle) probabilities, but there is no indication in the structure of

![Diagram](https://example.com/diagram.png)
The results of the previous mathematical formulation can now be obtained by fixed point iteration. In Fig. 3, we present the results for homogeneous random networks, when the simplicial structure is not considered ($\beta^\Delta = 0$), and when 2-simplices are included ($\beta^\Delta \neq 0$). We observe that in the second case, when the simplicial structure is considered for the higher-order dynamics of the SIS model, the incidence of the epidemics $\rho = \frac{1}{2} \sum P_i$ reveals an abrupt transition in all the previous approaches: MMCA, its mean-field version MF, and ELE. Nevertheless, the most accurate approximation when compared with the Monte Carlo simulation of the system, is provided by the epidemic link equations ELE. This is specially crucial when we try to capture the critical point of the transition. Unfortunately, the critical point at which the transition occurs eludes our analytical determination in ELE, given that nor the usual linearization technique [22,28], neither the next generation matrix method (NGM) [35] provided meaningful results. In Fig. 4, we corroborate our results on heterogeneous (scale-free) networks constructed following the analytical proposal in Ref. [34]. We observe how heterogeneity in degree highlights the differences in the determination of the critical point obtained by ELE, MMCA, and MF.

Summarizing, we have presented the mathematical formulation of the SIS model in simplicial complexes, using a discrete time probabilistic description of the process, in the node approximation MMCA, and in the link approximation ELE. Both descriptions predict an abrupt transition, as well as the stationary homogeneous approximation of the MMCA, the MF. The accuracy of the predictions is largely better for ELE, and reveals that this approximation is extremely useful when dealing with the simplicial geometry of complex networks. For the determination of the critical points of ELE, we think that further analysis using ideas on stability of subsystems [36] of 1-simplices and 2-simplices, are key components in the higher-order dynamics of the SIS model, the incidence on heterogeneous (scale-free) networks constructed following the numerical proposal in Ref. [34]. We observe how heterogeneity in degree highlights the differences in the determination of the critical point obtained by ELE, MMCA, and MF.

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References:
[1] S. H. Strogatz, *Sync: The Emerging Science of Spontaneous Order* (Hypernion, 2003).
[2] A. Arenas, A. Díaz-Guilera, J. Kurths, Y. Moreno, and C. Zhou, Synchronization in complex networks, *Phys. Rep.* 469, 93 (2008).
[3] A. Barrat, M. Barthélémy, and A. Vespignani, *Dynamical Processes on Complex Networks* (Cambridge University Press, New York, NY, USA, 2008).
[4] M. E. J. Newman, *Networks* (Oxford University Press, Oxford, New York, 2010).
[5] R. Pastor-Satorras, C. Castellano, P. Van Mieghem, and A. Vespignani, Epidemic processes in complex networks, *Rev. Mod. Phys.* 87, 925 (2015).
[6] Z. Wu, G. Menichetti, C. Rahmede, and G. Bianconi, Emergent complex network geometry, *Sci. Rep.* 5, 10073 (2015).
[7] V. Salnikov, D. Cassese, R. Lambiotte, Simplicial complexes and complex systems, *Eur. J. Phys.* **40**, 014001 (2019).
[8] D. Horak, S. Maletić, and M. Rajković, Persistent homology of complex networks, *J. Stat. Mech.* (2009) P03034.
[9] A. R. Benson, R. Abebe, M. T. Schaub, A. Jadbabaie, and J. Kleinberg, Simplicial closure and higher-order link prediction, *Proc. Natl. Acad. Sci. USA* **115**, E11221 (2018).
[10] N. Otter, M. A. Porter, U. Tillman, P. Grindrod, and H. A. Harrington, A roadmap for the computation of persistent homology, *Euro. Phys. J. DS* **6**, 17 (2017).
[11] G. Petri, P. Expert, F. Turkheimer, R. Carhart-Harris, D. Nutt, P. J. Hellyer, and F. Vaccarino, Homological scaffolds of brain functional networks, *J. R. Soc. Interface* **11**, 20140873 (2014).
[12] C. Giusti, R. Ghrist, and D. S. Bassett, Two’s company, three (or more) is a simplex, *J. Comput. Neurosci.* **41**, 1 (2016).
[13] P. Ashwin and A. Rodrigues, Hopf normal form with $S_N$ symmetry and reduction to systems of nonlinearly coupled phase oscillators, *Physica D* **325**, 14 (2016).
[14] M. W. Reimann, M. Nolte, M. Scalamiero, K. Turner, R. Perin, G. Chindemi, P. Dlotko, R. Levi, K. Hess, and H. Markram, Cliques of neurons bound into cavities provide a missing link between structure and function, *Front. Comp. Neuro.* **11**, 48 (2017).
[15] A. P. Millán, J. J. Torres, and G. Bianconi, Complex network geometry and frustrated synchronization, *Sci. Rep* **8**, 9910 (2018).
[16] A. P. Millán, J. J. Torres, and G. Bianconi, Synchronization in network geometries with finite spectral dimension, *Phys. Rev. E* **99**, 022307 (2019).
[17] I. Iacopini, G. Petri, A. Barrat, and V. Latora, Simplicial models of social contagion, *Nat. Commun.* **10**, 2485 (2019).
[18] P. S. Skardal and A. Arenas, Abrupt Desynchronization and Extensive Multistability in Globally Coupled Oscillator Simplexes, *Phys. Rev. Lett.* **122**, 248301 (2019).
[19] R. Lambiotte, M. Rosvall, and I. Scholtes, From networks to optimal higher-order models of complex systems, *Nat. Phys.* **15**, 313 (2019).
[20] H. W. Hethcote, The mathematics of infectious diseases, *SIAM Rev.* **42**, 599 (2000).
[21] R. D’Souza, J. Gómez-Gardeñes, J. Nagler, and A. Arenas, Explosive phenomena in complex networks, *Adv. Phys.* **68**, 123 (2019).
[22] S. Gómez, A. Arenas, J. Borge-Holthoefer, S. Meloni, and Y. Moreno, Discrete-time Markov chain approach to contact-based disease spreading in complex networks, *Europhys. Lett.* **89**, 38009 (2010).
[23] S. Gómez, J. Gómez-Gardeñes, Y. Moreno, and A. Arenas, Nonperturbative heterogeneous mean-field approach to epidemic spreading in complex networks, *Phys. Rev. E* **84**, 036105 (2011).
[24] C. Granell, S. Gómez, and A. Arenas, Dynamical Interplay between Awareness and Epidemic Spreading in Multiplex Networks, *Phys. Rev. Lett.* **111**, 128701 (2013).
[25] C. Granell, S. Gómez, and A. Arenas, Competing spreading processes on multiplex networks: Awareness and epidemics, *Phys. Rev. E* **90**, 012808 (2014).
[26] J. Gómez-Gardenes, D. Soriano-Panos, and A. Arenas, Critical regimes driven by recurrent mobility patterns of reaction-diffusion processes in networks, *Nat. Phys.* **14**, 391 (2018).
[27] D. Soriano-Panos, L. Lotero, A. Arenas, and J. Gómez-Gardenes, Spreading Processes in Multiplex Metapopulations Containing Different Mobility Networks, *Phys. Rev. X* **8**, 031039 (2018).
[28] J. T. Matamalas, A. Arenas, and S. Gómez, Effective approach to epidemic containment using link equations in complex networks, *Sci. Adv.* **4**, eaau4212 (2018).
[29] A. R. Lucas, A fixed point theorem for a general epidemic model, *I. Math. Anal. Appl.* **404**, 135 (2013).
[30] M. Henkel, H. Hinrichsen, and S. Lubeck, *Non-equilibrium Phase Transition: Absorbing Phase Transitions* (Springer Verlag, The Netherlands, 2008).
[31] A. S. Mata and S. C. Ferreira, Pair quenched mean-field theory for the susceptible-infected-susceptible model on complex networks, *Europhys. Lett.* **103**, 48003 (2013).
[32] E. Cator and P. VanMieghem, Second-order mean-field susceptible-infected-susceptible epidemic threshold, *Phys. Rev. E* **85**, 056111 (2012).
[33] S. C. Ferreira, C. Castellano, and R. Pastor-Satorras, Epidemic thresholds of the susceptible-infected-susceptible model on networks: A comparison of numerical and theoretical results, *Phys. Rev. E* **86**, 041125 (2012).
[34] S. N. Dorogovtsev, A. V. Goltsev, and J. F. F. Mendes, Pseudofractal scale-free web, *Phys. Rev. E* **65**, 066122 (2002).
[35] O. Diekmann, J. A. P. Heesterbeek, and J. A. J. Metz, On the definition and computation of the basic reproduction ratio $R_0$ in models for infectious diseases in heterogeneous populations, *J. Math. Biol.* **28**, 365 (1990).
[36] M. Perc, Stability of subsystem solutions in agent-based models, *Eur. J. Phys.* **39**, 014001 (2017).