Dynamics of epidemic models from cavity master equations

Ernesto Ortega, David Machado, Alejandro Lage-Castellanos

We apply the cavity master equation (CME) approach to epidemics models. We explore mostly the susceptible-infectious-susceptible (SIS) model, which can be readily treated with the CME as a two-state. We show that this approach is more accurate than individual based and pair based mean field methods, and a previously published dynamic message passing scheme. We explore average case predictions and extend the cavity master equation to SIR and SIRS models.

I. INTRODUCTION

Since the seminal works introducing susceptible-infectious-recovered compartment models (SIR) of Kermack and McKendric [1], epidemics modeling has grown fast as a field. The approach has changed with time, from dynamical systems or population dynamics towards a more stratified approaches as patchy, mobility based, age-structured or contact matrices compartment models.

The current context of a global COVID-19 outbreak and the perspective of a coexistence with an endemic virus requires a test-trace-isolate epidemiological system to keep the outbreak controlled. Much attention is now put on agent based models that could improve the efficacy of the testing strategy. Assuming that new technologies can provide reliable contact data between humans, the likelihood of people being infected needs to be estimated either by numerical simulations or some statistical modeling. To this end, it is suitable to count with fast algorithms that can accurately predict probabilities of infection for agents in networks.

There are a wide variety of such algorithms. The classical approach to the forecasting of epidemics on networks is an averaging of the master equation of the process complemented by a factorization assumption at some level. This yields a hierarchy of ever more complex but more accurate differential equations for expected values and correlations [2, 3]. Most of the time only the first two levels, known as individual based mean field and pair based mean field, are used.

More recently, ideas from discrete optimization algorithms have sneaked into the inference of SIR kind of models in the shape of Dynamical Message Passing [4, 5] or Belief Propagation (BP) [6, 7]. The main difference with respect to the previous approach is the appearance of conditional probabilities, rather than correlations, to be integrated in time. It has been used with success in the reconstruction of epidemics on graphs and it is currently being tried in the task of risk assessment for COVID-19.

It is known that BP’s fixed point is connected to the cavity method from statistical mechanics [8]. An extension of the latter to continuous time Markov Chain processes within discrete spin systems have been recently achieved through the derivation of a set of differential equations for cavity conditional probabilities: the cavity master equation (CME) [9].

In this article we explore CME’s application to SIS and SIR-like models in graphs (section III). When compared with Monte Carlo simulations of the epidemic, it is shown to outperform three other approximations. In some simple cases we draw analytical results for the steady state and critical reproduction number (section IV). We also explore the average case dynamics of these equations, in particular in random regular and Erdos-Renyi graphs (sec V). We conclude by briefly showing the application of CME to SIR and SIRS models in section VI.

II. EPIDEMICS ON NETWORKS

In what follows we focus on continuous time compartmental epidemic models on networks. We assume a fixed network of contacts to be given $G = (V, E)$ with a set of vertices $V = \{1, 2, \ldots, N\}$ and a set of edges $E$. An edge $(i, j)$ is present if nodes $i$ and $j$ are neighbors in the network, meaning there is a possibility of transmission of diseases between both nodes.

The standard susceptible-infectious-susceptible model (SIS) considers the nodes to be in either of two compartments (states) $X_i = 0 \equiv$ susceptible, or $X_i = 1 \equiv$ infectious and is the simplest standard for recurrent transmissible diseases. The epidemic is thus a continuous time stochastic process with only two admitted transitions occurring at

- a rate $\beta$, at which a link $(i, j)$ can transmit the state 1 from node $i$ to $j$;
- a rate $\mu$ at which state 1 decays to state 0 on any infectious node.

as represented in diagram 1. An analytical description of this stochastic process is given by the master equation for the evolution in time of the probability over the whole configuration space $P(X_1, \ldots, X_N, t)$ [3]. However concise and exact, the integration in time of such equation is generally impractical given the size $2^N$ of the configuration space.
Attempts to reduce the complexity start from factorizing the single master equation into many equations for each node marginals \( P_i(X_i, t) \). Given that the \( X \)'s are two state variables, \( P_i(X_i) \) is parameterized by the mean value \( E[X_i] = P_i(X_i = 1) \). This results in an equation that is still exact [3]

\[
\frac{dE[X_i(t)]}{dt} = E \left[ -\mu X_i(t) + (1 - X_i(t)) \beta \sum_{j=1}^{N} a_{ij} X_j(t) \right]
\]

where \( a_{ij} = 1 \) if \( (i, j) \in E \) and is zero if nodes \( i \) and \( j \) are not neighbors in the graph.

However, the expectation value on the right hand side acts over products of variables \( X_i(t)X_j(t) \), which requires a differential equation for the evolution of the correlations. Not surprisingly, the two point correlations functions depend on three point correlations and so on and so forth.

The simplest closure of equation eq. (1) is the individual-based mean field (IBMF) in which independence is assumed

\[
E[X_i(t)X_j(t)] = E[X_i(t)] E[X_j(t)] = \rho_i(t) \rho_j(t)
\]

and therefore eqs. (1) are now a closed set of non linear differential equations:

\[
\frac{d\rho_i(t)}{dt} = -\rho_i(t) + \lambda[1 - \rho_i(t)] \sum_{j=1}^{N} a_{ij} \rho_j(t)
\]

The second simplest closure is that known as pair-based mean field (PBMF), in which two point correlations are treated analytically [10]:

\[
\frac{dE[X_iX_j]}{dt} = -2\mu E[X_iX_j] + \beta \sum_{k=1}^{N} a_{ik} E[X_iX_k] + \beta \sum_{k=1}^{N} a_{jk} E[X_iX_k] - \beta \sum_{k=1}^{N} (a_{ik} + a_{jk}) E[X_iX_jX_k]
\]

but a factorization is assumed for higher correlations. Different approaches have been used to approximate \( E[X_iX_jX_k] \) in terms of smaller correlations. We will compare to that in which \( E[X_iX_jX_k] \equiv E[X_iX_j] E[X_k] \equiv \rho_{ij}(t) \rho_k(t) \)

\[
\frac{dp_{ij}(t)}{dt} = -\mu p_{ij}(t) + \beta \sum_{j'} [p_{ij'}(t) - p_{ij}(t)]
\]

\[
\frac{dp_{ij}(t)}{dt} = -2\mu p_{ij}(t) + \beta [p_i(t) + p_j(t) - 2p_{ij}(t)] + \beta \sum_{k \in \partial i \setminus j} [p_{ik}(t) - p_{ij}(t)p_k(t)] + \beta \sum_{k \in \partial j \setminus i} [p_{ik}(t) - p_{ij}(t)p_k(t)]
\]

In both approaches, IBMF and PBMF, the expected values evolving in time are intended to be expectations over different stochastic stories of the whole epidemic process. Therefore they are to be compared with averages over many Monte Carlo simulations of such process.

A slightly different approach to modeling epidemics on graphs come from message-passing inspired methods. The dynamic message passing [4, 5] involves a set of probabilities and a set of conditional probabilities, rather than correlations [5]:

\[
\frac{dp_i}{dt} = -\mu p_i + \beta(1 - p_i) \sum_k p_{ki}
\]

\[
\frac{dp_{ij}}{dt} = -\mu p_{ij} + (1 - p_j) \beta \sum_{k \in \partial i \setminus j} p_{ki}
\]
where \( p_i = \text{E}[X_i] \) just as before, but \( p_{ij} \equiv P(X_i = 1 | X_j = 0) \) is a conditional probability resembling the kind of cavity fields or messages that commonly appear in the cavity method and the belief propagation algorithm for discrete optimization.

The main results of this article are drawn from the cavity master equation \([9]\). They are quite similar to those of rDMP and help formalizing this rather empirical approach by deriving it from a more solid mathematical setting. In doing so we not only correct one term of the rDMP equations, but also underline the approximations involved and therefore shed light on possible improvements.

### III. CAVITY MASTER EQUATIONS FOR SIS EPIDEMICS

In order to connect with its first presentation in \([9]\), we start by considering the general continuous time dynamics of a system \( \sigma = \{\sigma_1, \ldots, \sigma_N\} \) of \( N \) bimodal variables \( \sigma_i \in \{\pm 1\} \) interacting with their nearest neighbors in some given topology. In the very generic Markovian case, the dynamic is fully defined by the rate function \( r_i(\sigma) \) at which variables flip their states from \( \sigma_i \to -\sigma_i \). The distribution \( P(\sigma, t) \) in the configuration space of this stochastic process is ruled by the joint master equation:

\[
\frac{dP(\sigma)}{dt} = -\sum_{i=1}^{N} \left[ r_i(\sigma)P(\sigma) - r_i(F_i(\sigma))P(F_i(\sigma)) \right],
\]

where \( F_i \) represents the flip operator on variable \( i \), i.e. \( F_i(\sigma) = \{\sigma_1, \ldots, \sigma_{i-1}, -\sigma_i, \sigma_{i+1}, \ldots, \sigma_N\} \).

Although exact, the previous equation is useless already for middle size systems, since it actually represents a set of \( 2^N \) coupled differential equations that take exponential time to enumerate, let alone to integrate. However this is the correct starting point for approximations, as is usually done to obtain mean field approximations (2,4,5) in epidemics models \([2, 3, 11]\).

In \([9]\) this master equation is recast into an equilibrium problem by extending the configuration space to consider the continuous trajectory of each variable in time \( X = \{X_1, \ldots, X_N\} \) where \( X_i = \{\sigma_i(t) : \forall t \in [0, T]\} \). Although at glance it seems untreatable the infinite dimensional space for the functions \( X_i(t) \), the discrete values of \( \sigma_i(t) \) allows for a codification of the functions in a numerable number of transitions times \( \{T_1^{(i)}, T_2^{(i)}, \ldots\} \) such that \( \sigma_i(T_k^{(i)}) = -\sigma_i(T_k^{(i)} + dt) \). The resulting Random Point Process is treated with standard techniques in equilibrium statistical mechanics to write down a closed set of cavity master equations as:

\[
\frac{dP(\sigma_i)}{dt} = -\sum_{\sigma_{i\neq j}} \left[ r_i(\sigma_i, \sigma_{i\neq j}) \left( \prod_{k \in \partial i} p(\sigma_{k|\sigma_i})P(\sigma_i) - r_i(-\sigma_i, \sigma_{i\neq j}) \left( \prod_{k \in \partial i} p(\sigma_{k|\sigma_i} - \sigma_i) \right) P(-\sigma_i) \right]
\]

\[
\frac{dp(\sigma_i|\sigma_j)}{dt} = -\sum_{\sigma_{i\neq j}} \left[ r_i(\sigma_i, \sigma_{i\neq j}) \left( \prod_{k \in \partial i \setminus j} p(\sigma_{k|\sigma_i})\right) p(\sigma_i|\sigma_j) - r_i(-\sigma_i, \sigma_{i\neq j}) \left( \prod_{k \in \partial i \setminus j} p(\sigma_{k|\sigma_i} - \sigma_i) \right) p(-\sigma_i|\sigma_j) \right]
\]

We have lighten the notation by not putting the \( i \) and \( ij \) dependence of the distributions, understanding that they assume the index of the variables they depend on (as \( P(\sigma_i) \equiv P(\sigma_i) \)). Both probabilities are intended in the sense “over the ensemble of dynamic evolutions up to time \( t \)”, starting from the same initial conditions.

This is a substantial improvement over the original master equation since we are dealing now with \( N \) functions \( P_i(\sigma_i, t) \) representing the distribution of variables \( \sigma_i \), and with \( N \times \langle k \rangle \) functions \( P_{i\langle k \rangle}(\sigma_i|\sigma_{j\langle k \rangle}) \) that represent the probability of finding variables in a given state, conditioned to the state of one of its neighbors (\( \langle k \rangle \) is the average degree of the nodes in the interactions network). In the worst case of a fully connected system, we still would have \( O(N^2) \) equations, that can be numerically integrated even for relatively large systems, compared to what we can do with eq. (8).

We can readily translate the cavity master equations (9,10) to the case of S.I.S epidemic model by identifying our two states as \( S \to \sigma_i = -1 \) and \( I \to \sigma_i = 1 \). After complementary \( P(I, I) + P(S, S) = 1 \), it is enough to track the infection probability in each node \( P_i(I) \). The first term in equation (9) largely simplifies due to the fact that the recovery rate \( r_i(1, -\sigma_i) = \mu \) is independent of the neighbors state, resulting in \( -\mu P(I) \). Considering also that the transmission rates are additive \( r_i(S, \sigma_{i\neq j}) = \sum_{k'} r_i(S, \sigma_{k'}) \) and that \( r_i(S, S) = 0 \) and \( r_i(S, I) = \beta \) we get to the cavity master equations for the S.I.S model as

\[
\frac{dP_i}{dt} = -\mu p_i + \beta (1 - p_i) \sum_k p_{ki}
\]

\[
\frac{dp_{ij}}{dt} = -\mu p_{ij} + (1 - p_{ij}) \beta \sum_{k \in \partial i \setminus j} p_{ki}
\]
Fig. 2. **Left:** Probability of node 29 to be infected as a function of time, discounting at each time the situations in which the epidemics disappear. The epidemic outbreak was in node 1, with $\beta = 0.1$ and $\mu = 0.05$. The inset is a closer look in the stationary state. **Right:** L1 distance between the four approximations IBMF, PBMF, rDMP, CME with respect to MC simulations.

where we simplified notation further by making $P(\sigma_i = 1) \equiv p_i$ and $P(\sigma_i = 1 | \sigma_j = -1) \equiv p_{ij}$. Appendix A contains a more detailed derivation of these equations for SIS model.

Equations (11) and (12) are almost identical to those obtained for rDMP (6) and (7), except for the very last term where $(1 - p_j)$ is replaced by $(1 - p_{ij})$. Not surprisingly, the results from both approximations might not be too different in certain cases. For instance, by reproducing the very same case of [5] of an SIS epidemic outbreak in the Zacharia’s karate club network, starting at node 1 both approaches are quite similar as shown in figure 7 (left) (actually, in that figure is almost impossible to distinguish one from the other and CME seems to be missing). In figure 7 (right) the L1 distance between the average from many Monte Carlo simulations and the predictions made by all four methods CME, rDMP, IBMF and PBMF shows features that will repeat in other benchmarks:

- that both rDMP and CME get the general qualitative behavior well,
- with a rather faster outbreak expansion in the transient and
- with CME better fitting the stationary state.

Individual based mean field tends to be the fastest growing prediction, while pair based mean field is the closest to CME, yet not equal to it.

When computing the Monte Carlo averages we have neglected the simulations in which the epidemic is randomly wiped out in the first few iterations. None of these methods can take this fluctuations into account, since they are all mean-field approaches.

Differences are more evident in the case of random regular graphs with degree $k = 3$ in figure 3. We represent the average epidemic size, i.e. the fraction of infected nodes respect to number $N = 1000$ of nodes in the graph at every time step. We started from a fraction $\alpha = 0.5$ of nodes infected, and observed the onset of the endemic state.

### IV. ENDEMIC (STEADY) STATE

We can easily compute the epidemic size in the endemic state for these approximations in the case of random regular graphs. Since every node has the same amount of neighbors $k$, the equations are the same for every node, and we can assume that in the steady state the topology is averaged out, and all the probabilities are the same, regardless the node indexes.

Working explicitly for the CME approximation, we set $\frac{dp_i}{dt}$ and $\frac{dp_{ij}}{dt}$ to 0 in equations (11) and (12):

$$\mu p_i = \beta (1 - p_i) \sum_k p_{ki} \equiv \beta (1 - p_i) \beta \sum_{k \in \partial i \setminus j} p_{ki} \equiv (1 - p_{ij}) \beta (k - 1)$$

where now the indices $i$ and $i, j$ are generic. Solving this system of equations we get for the two variables $p_i$ and $p_{ij}$:

$$p_i = \frac{1}{1 + \frac{\mu}{(k - 1)\beta}}$$

$$p_{ij} = 1 - \frac{\mu}{(k - 1)\beta}$$

(14)
![Random regular graph with connectivity k = 3. Top figures: Percent of the population in the infected state (left) and the $L_1$ distance (right), in a graph of 100 nodes with parameters $\beta = 0.11$ and $\mu = 0.1$ and an outbreak in one node of the system. Bottom figures: Percent of the population in the infected state for different values $Ro$ in a graph of 1000 nodes, starting with half of the population in the infected state.](image)

This can be already tested against simulations of random regular graphs. Furthermore we can obtain the critical reproduction number $R_0 = \beta/\mu$ above which there is an endemic outbreak (sustained in time epidemic) by solving $p_i(k) = 0$, resulting in $R_0^* = 1/(k - 1)$. A similar procedure for all four approximations results in table I.

In figure 4 we present the analytical predictions of each approximation for the epidemic size $p_i$ at the steady state as a function of $R_0$. When compared to Monte Carlo results, mean field approximations (IBMF and PBMF) give an overestimation of the epidemic size at small $R_0$, and rDMP an underestimation at large $R_0$. Meanwhile, CME seems to be a better prediction mixing the large $R_0$ behavior of IBMF and PBMF with the small $R_0$ behavior of rDMP.

V. AVERAGE CASE

The systems of equations defining IBMF, PBMF, rDMP and CME could be large and delicate to solve, although a simple numerical integration normally works. However, in many cases we are interested in general predictions for certain families of graphs or topologies. In this section we derive an average version of the CME approximation to characterize SIS epidemics on random graphs.

The simplest description of a graph ensemble is given by the distribution of degrees of its nodes. Since the CME equations depend on the information coming from the neighbors in the network, it is expected that nodes more connected will have a different behavior than those less connected. We therefore attempt to reduce the number of equations in our system by characterizing all nodes with the same degree by a couple of average parameters

$$p^\gamma = \frac{1}{N^\gamma} \sum_{i:d_i = \gamma + 1} p_i, \quad p^{\gamma}_{ij} = \frac{1}{M^\gamma} \sum_{i:d_i = \gamma + 1} \sum_{j \in \partial i} p^\gamma_{ij}$$

(15)
Approximation | Endemic state (equilibrium) | $R_0^*$
---|---|---
IBMF | $p_i = 1 - \frac{k}{k\lambda}$ | $

PBMF | $p_i = \frac{(R_0)^2 k + R_0 - (\frac{1}{k\lambda})}{(R_0)^2 k + 2R_0}$ | $R_0^* = \sqrt{\frac{1 + f_k}{2k} - 1}$

rDMP | $p_i = 1 - \frac{\mu}{(k - 1)\beta}$ | $p_{ij} = 1 - \frac{\mu}{(k - 1)\beta}$ | $R_0^* = \frac{1}{k - 1}$

CME | $p_i = \frac{1}{1 + \frac{k\beta}{(k - 1)\beta - \mu}}$ | $p_{ij} = 1 - \frac{\mu}{(k - 1)\beta}$ | $R_0^* = \frac{1}{k - 1}$

**TABLE I.** Critical reproduction number under four approximations: IBMF, PBMF, rDMP and CME

![Graphs showing comparisons between Monte Carlo simulations and the predicted endemic states of each method as a function of $R_0$. Right: Comparison between the epidemic threshold predicted by the methods as a function of graphs connectivity.](image)

In both cases the normalization factors count the number of terms in the sums: $N^\gamma$ is the number of nodes with connectivity $\gamma + 1$ while $M^\gamma$ is the number of graph’s edges that contain one of these $N^\gamma$ nodes. Averaging the equations (11) and (12), and after some simplifications, we get the average CME:

$$\dot{p}^\gamma = -\mu p^\gamma + \beta(\gamma + 1)(1 - p^\gamma) \sum_{\gamma'} g_{\text{link}}(\gamma') p^\gamma_{\gamma'}$$

$$\dot{p}^\gamma_{\gamma'} = -\mu p^\gamma_{\gamma'} + \beta(1 - p^\gamma_{\gamma'}) \sum_{\gamma''} g_{\text{link}}(\gamma'') p^\gamma_{\gamma''}$$

where $g(\gamma)$ is a contact degrees distribution. A node extracted randomly from the set of nodes $V$ has degree distribution $P(\gamma)$. However, in order to average the CME equations we rather need to know the degree distribution $g(\gamma)$ of nodes that are sampled by randomly picking up an edge $(i, j) \in E$. Both distributions are related, assuming there is no further correlations in the graph ensemble, by:

$$g_{\text{link}}(\gamma) = \frac{(\gamma + 1)P(\gamma)}{\sum_\gamma (\gamma + 1)P(\gamma)}$$

A very simple case is that of regular graphs, where degree distribution is deltaic $P(\gamma) = g(\gamma) = \delta_{k-1, \gamma}$. Equations
Fig. 5. Epidemic size as function of time. The epidemic outbreak was in 5 nodes of the graph of 100 nodes and connectivity 5 for each node, with $\beta = 0.1$ and $\mu = 0.05$.

Equations (16) and (17) reduce to two equations for the parameters $p^{k-1}(t) \equiv p(t)$ and $p^{\gamma \rightarrow}(t) \equiv p_{\rightarrow}(t)$:

$$
\dot{p} = -\mu \tilde{p} + \beta k \tilde{p}(1-p) \\
\dot{p}_{\rightarrow} = -\mu \tilde{p}_{\rightarrow} + \beta (k-1) \tilde{p}_{\rightarrow}(1-p_{\rightarrow})
$$

whose numerical integration can be compared with Monte Carlo simulations of epidemics in graphs with the same vertex degree $k$, and with the corresponding integration of the single instance CME equations (11) and (12). Figure 5 shows that the steady state is well predicted, while the transient is not, even in comparison with CME itself. This is a natural consequence of the lost of the spatial structure in the average case.

A. General graph ensembles

Equations (16) and (17) are already a reduction of $N$ differential equations to $K$ average equations, where $K$ is the maximum degree in the graph. However, $K$ itself could be large. We can further simplify by averaging now over the nodes degree and reducing to only two parameters $\tilde{p}_{\rightarrow} = \sum_{\gamma} g_{\text{link}}(\gamma)p_{\rightarrow}^{\gamma}$, and $\tilde{p} = \sum_{\gamma} P(\gamma)p^{\gamma}$:

$$
\dot{\tilde{p}} = -\mu \tilde{p} + \beta \tilde{p}_{\rightarrow} \sum_{\gamma} (\gamma + 1)P(\gamma)(1-p^{\gamma})
$$

$$
\dot{\tilde{p}}_{\rightarrow} = -\mu \tilde{p}_{\rightarrow} + \beta \tilde{p}_{\rightarrow} \sum_{\gamma} \gamma g_{\text{link}}(\gamma)(1-p_{\rightarrow}^{\gamma})
$$

These equations are still not closed, since the right hand sides still depend on the degree based parameters. The product by $(\gamma + 1)$ and $\gamma$ inside the sums in the right hand sides do not allow for a direct connection with the definitions of $\tilde{p}$ and $\tilde{p}_{\rightarrow}$. We will show, however, that in the case of Erdos-Renyi graphs such connection can be obtained, though through some approximations and ansatzs.

B. Closure on Erdos Renyi graphs

For an Erdos-Renyi graph node degrees are Poisson-distributed: $P(\gamma) = \frac{e^{-\kappa} \kappa^{\gamma+1}}{(\gamma+1)!}$ and $g_{\text{link}}(\gamma) = P(\gamma - 1)$, where $\kappa$ is the average degree. We can connect the terms inside the sums in (20) and (21) with the derivatives of $\tilde{p}$ and $\tilde{p}_{\rightarrow}$ with respect to the parameter $\kappa$ by noting that

$$
\frac{\partial \tilde{p}}{\partial \kappa} = -\tilde{p} + \frac{1}{\kappa} \sum_{\gamma} (\gamma + 1)P(\gamma)p^{\gamma}
$$

$$
\frac{\partial \tilde{p}_{\rightarrow}}{\partial \kappa} = -\tilde{p}_{\rightarrow} + \frac{1}{\kappa} \sum_{\gamma} \gamma g_{\text{link}}(\gamma)p_{\rightarrow}^{\gamma}
$$
Fig. 6. Comparison between average-CME and CME for Erdos Renyi graphs with mean connectivity $c_0 = 3$, several values of $\mu$ and $\beta = 0.4$. The figure shows the single site probability of infection as a function of time. As the ratio $\beta/\mu$ decreases, the steady state has less infection probability.

As it is shown in appendix B, by substitution in (20) and (21) we get:

$$\dot{\tilde{p}} = -\mu \tilde{p} + \beta \kappa \tilde{p}_{\to} \left[ 1 - \tilde{p} - \frac{\partial \tilde{p}_{\to}}{\partial \kappa} \right]$$  \hspace{1cm} (24)

$$\dot{\tilde{p}}_{\to} = [\beta \kappa - \mu] \tilde{p}_{\to} - \beta \kappa \tilde{p}_{\to} \left( \tilde{p}_{\to} + \frac{\partial \tilde{p}_{\to}}{\partial \kappa} \right)$$  \hspace{1cm} (25)

In order to solve these equations we need some ansatz for the dependence of the mean values $\tilde{p}$ and $\tilde{p}_{\to}$ on the mean degree in the graph. Inspired by the whole derivation of the Cavity Master Equation for spin systems, we propose

$$\tilde{p}(t) = \frac{1}{2} [1 + \tanh (\beta \kappa \chi(t) - \mu)]$$  \hspace{1cm} (26)

$$\tilde{p}_{\to}(t) = \frac{1}{2} [1 + \tanh (\beta \kappa \epsilon(t) - \mu)]$$  \hspace{1cm} (27)

where $\chi(t)$ and $\epsilon(t)$ are some time-dependent fields that are obtained by inverting these very formulas in terms of $\tilde{p}$ and $\tilde{p}_{\to}$.

From this ansatz we can express the derivatives with respect to the degree in (24) and (25) in terms of $\tilde{p}$ and $\tilde{p}_{\to}$, respectively (see appendix B). We get then a closed system of differential equations for these probabilities, that can be solved numerically. The results of the integration are shown in figure 6.

VI. EXTENSIONS TO SIR-SIRS MODELS

Models with more compartments are also ubiquitous in epidemics modeling. In particular the SIR family, including SEIR and SIRS, that have experienced a boost in attention due to the COVID-19 currently ongoing epidemic. The Cavity Master Equation, that was introduced for spins $\sigma_i = \pm 1$ physical models, could be translated with some effort to multi states models, like Potts $q$-states models or SIR. We leave such general and formal approach to be attempted in a separate article. Instead, we will just extrapolate the type of equations obtained from spin variables to $q$-state variables, and briefly compare with simulations to show its plausibility for SIR and SIRS.
A generalization of eqs. (9) and (10) to models with q states is proposed as

\[
\frac{dP_i(\sigma_i)}{dt} = \sum_{\sigma'_i} \frac{(-1)^{\delta_{\sigma_i, \sigma'_i}} r_i(\sigma_i', \sigma_{\partial_i}) [\prod_{k \in \partial_i} P_{ki}(\sigma_k|\sigma')] P_i(\sigma'_i)}{P_i(\sigma'_i)}
\]

(28)

\[
\frac{dp_{ij}(\sigma_i, \sigma_j)}{dt} = \sum_{\sigma'_i, \sigma'_{\partial_i,j}} \frac{(-1)^{\delta_{\sigma_i, \sigma'_i}} r_{ij}(\sigma_i', \sigma_j', \sigma_{\partial_i,j}) [\prod_{k \in \partial_i,j} p_{ki}(\sigma_k|\sigma')] p_{ij}(\sigma'_i|\sigma_j)}{p_{ij}(\sigma'_i|\sigma_j)}
\]

(29)

Sums run over all q states of each variable (q \in \{S, I, R\} for SIR and SIRS), and the (−1) factor chooses between terms that contribute to the state \(\sigma' = \sigma_i\), and terms that take \(\sigma_i\) to some other configuration \(\sigma'_i\). For instance, in the SIR and SIRS cases the equations reduce to (after considering the explicit form of the rate functions):

\[
\frac{dP_i(\sigma_i \equiv I)}{dt} = -\mu P_i(I) + \beta P_i(S) \sum_{k \in \partial_i} p_{ki}(I|S)
\]

(30)

\[
\frac{dP_i(S)}{dt} = \gamma P_i(R) - \beta P_i(S) \sum_{k \in \partial_i} p_{ki}(I|S)
\]

(31)

\[
\frac{dP_i(R)}{dt} = -\gamma P_i(R) + \mu P_i(I)
\]

(32)

\[
\frac{dp_{ij}(I|S)}{dt} = -\mu p_{ij}(I|S) + \beta p_{ij}(S|S) \sum_{k \in \partial_i,j} p_{ki}(I|S)
\]

(33)

\[
\frac{dp_{ij}(S|S)}{dt} = \gamma p_{ij}(R|S) - \beta p_{ij}(S|S) \sum_{k \in \partial_i,j} p_{ki}(I|S)
\]

(34)

\[
\frac{dp_{ij}(R|S)}{dt} = -\gamma p_{ij}(R|S) + \mu p_{ij}(I|S)
\]

(35)

Constants \(\mu\) and \(\beta\) are still the recovery rate and the infection rate, while \(\gamma\) is the rate at which immunity is lost and patients pass from the recovered compartment back into the susceptible one. In the case of SIRS model \(\gamma > 0\), while SIR corresponds to \(\gamma = 0\).

The first three equations (30,31,32) are still the same obtained by the dynamical message passing in [5] but, as in the SIS case, the conditional distribution equation (33) differs from that in rDMP

\[
\frac{dp_{ij}(I|S)}{dt} = -\mu p_{ij}(I|S) + \beta P_j(S) \sum_{k \in \partial_i,j} p_{ki}(I|S)
\]

(36)

in the factor \(P_j(S)\) before the sum over neighbors. Notice that this difference implies that only four equations are needed in the rDMP case: 3 for each node \(P_i(I), P_i(S), P_i(R)\) and one on each directed edge \(p_{ij}(I|S)\); while in our case all 6 equations need to be integrated (3 on nodes, 3 on edges) since they are all mutually dependent.

Numerical experiments show that the CME approach fits better the average evolution of Monte Carlo simulations (figure 7) for SIRS and SIR models.

VII. CONCLUSIONS

The analytical and numerical results on susceptible-infectious-susceptible model have shown that cavity master equation (CME) is an effective approximation to the average dynamics of epidemic systems, where average is intended over many stochastic realizations of the epidemics process. It outperforms individual and pair based mean field approximations, and corrects a term in the dynamic message passing approach. We have explored with some success the average case (topology) predictions for random regular and Erdos-Renyi graphs, and also showed that the approach is extensible to more elaborated models as SIR and SIRS.

Many aspects remain to be studied. First, although not shown, we have seen that CME (and probably also all others methods) fails when collective behavior appears, for instance, in the edge of the endemic transition. This is expected since a key step of the cavity master equation is the assumption of some sort of factorization. What is more
the transition rate from contact with an infected one (\(r\) or die) from the disease, so this rate is directly the recovering rate does not depends on the state of the contacts of the Infected person. It just depends on the typical time of recovering.

The rate \(r\) of the terms. Let’s take cases seems an appealing direction to us. The general case, in the graphs sense and also in the model sense (SIR, SIRS, SEIR), remains to be studied. Whether we can turn CME equations into some sort of jacobian stability approach for a general type of graphs seems also a good approach.

Third, the extension of CME to Potts models with \(q\) states need to be formalized in the framework of point processes. In this appendix we will present more details on the formulation of the CME for the SIS model. Let’s take as \(\beta = 0.1\) and \(\gamma = 0.05\) and both inserted graphics show the corresponding percentage of nodes in the recovered state.

surprising is that the approach is particular bad in 1D ring networks. Exploring why and how to improve in such cases seems an appealing direction to us.

Second, the average case solution is far from general, relying in some physical intuition to close the set of equations. The general case, in the graphs sense and also in the model sense (SIR, SIRS, SEIR), remains to be studied. Whether we can turn CME equations into some sort of jacobian stability approach for a general type of graphs seems also a good approach.

Third, the extension of CME to Potts models with \(q\) states need to be formalized in the framework of point processes that was used to derive CME in the first place. While doing this, attention should be put on the possibility of writing a CME-likelihood that could be used for inverse problems or for inference with partial information over some nodes, which is a key issue in current projects for testing and tracing.

Appendix A: Cavity master equation for SIS model.

In this appendix we will present more details on the formulation of the CME for the SIS model. Let’s take as starting points equations (9) and (10):

\[
\frac{dP(\sigma_i)}{dt} = - \sum_{\sigma_{\partial i}} \left[ r_i(\sigma_i, \sigma_{\partial i}) \prod_{k \in \partial i} P(\sigma_k | \sigma_i) P(\sigma_i) - r_i(-\sigma_i, \sigma_{\partial i}) \prod_{k \in \partial i} P(\sigma_k | -\sigma_i) P(-\sigma_i) \right]
\]

(A1)

\[
\frac{dp(\sigma_i)}{dt} = - \sum_{\sigma_{\partial i \cup}} \left[ r_i(\sigma_i, \sigma_{\partial i \cup}) \prod_{k \in \partial i \cup} p(\sigma_k | \sigma_i) p(\sigma_i | \sigma_j) - r_i(-\sigma_i, \sigma_{\partial i \cup}) \prod_{k \in \partial i \cup} p(\sigma_k | -\sigma_i) p(-\sigma_i | \sigma_j) \right]
\]

(A2)

Due to the complementarity of the probabilities \(P_i(I)\) and \(P_i(S)\) we can focus on just deriving the equation for one of the terms. Let’s take \(P_i(\sigma \equiv I)\) and rewrite equation (9) accordingly.

\[
\frac{dP_i(I)}{dt} = - \sum_{\sigma_{\partial i}} \left[ r_i(I, \sigma_{\partial i}) \prod_{k \in \partial i} P_{ki}(\sigma | I) P_i(I) - r_i(S, \sigma_{\partial i}) \prod_{k \in \partial i} P_{ki}(\sigma | S) P_i(S) \right]
\]

(A3)

The rate \(r_i(I, \sigma_{\partial i})\) is the transition rate from state \(I \rightarrow S\) and in the SIS model and in most of epidemic models it does not depends on the state of the contacts of the Infected person. It just depends on the typical time of recovering (or die) from the disease, so this rate is directly the recovering rate \(r_i(I, \sigma_{\partial i}) = \mu\). On the other hand \(r_i(S, \sigma_{\partial i})\) is the transition rate from \(S \rightarrow I\). In infectious diseases, the contagion can only occur if a susceptible individual is in contact with an infected one \((r_i(S, \sigma_k \equiv I) = \beta, r_i(S, \sigma_k \equiv S) = 0)\), and the probability of getting infected is additive with the number of infected contacts. This means that we can rewrite \(r_i(S, \sigma_{\partial i}) = \sum_{k \in \partial i} r_i(S, \sigma_k)\).

\[
\frac{dP_i(I)}{dt} = - \mu P_i(I) \sum_{\sigma_{\partial i}} \prod_{k \in \partial i} P_{ki}(\sigma | I) + P_i(S) \sum_{\sigma_{\partial i}} \left[ \sum_{k' \in \partial i} r_i(S, \sigma_{k'}) \prod_{k \in \partial i} P_{ki}(\sigma_k | S) \right]
\]

(A4)
Let’s apply the equivalence \( \sum \sigma_k \prod_{k \in \partial i} \rightarrow \prod_{k \in \partial i} \sum \sigma_k \) to invert the sum and product in the first term of the right hand side (RHS).

\[
\frac{dP_i(I)}{dt} = -\mu P_i(I) \prod_{k \in \partial i} \left[ \sum_{\sigma_k} P_ki(\sigma|I) \right] + P_i(S) \sum_{\sigma_k} \left[ \sum_{k' \in \partial i} r_i(S, \sigma_{k'}) \right] \prod_{k \in \partial i} P_ki(\sigma_k|S)
\] (A5)

The term \( \sum \sigma_k P_ki(\sigma|I) \) is exactly the sum of \( P_ki(I|I) + P_ki(S|I) = 1 \) and therefore also the product is equal to 1.

\[
\frac{dP_i(I)}{dt} = -\mu P_i(I) + P_i(S) \sum_{\sigma_k} \left[ \sum_{k' \in \partial i} r_i(S, \sigma_{k'}) \right] \prod_{k \in \partial i} P_ki(\sigma_k|S)
\] (A6)

Now we rewrite the second term of the RHS as follows:

\[
\frac{dP_i(I)}{dt} = -\mu P_i(I) + P_i(S) \sum_{\sigma_k} \sum_{k' \in \partial i} r_i(S, \sigma_{k'}) P_{k'\mid i}(\sigma|S) \prod_{k \in \partial i \setminus k'} P_ki(\sigma|S)
\] (A7)

\[
\frac{dP_i(I)}{dt} = -\mu P_i(I) + P_i(S) \sum_{\sigma_{k'}} \sum_{k' \in \partial i} r_i(S, \sigma_{k'}) P_{k'\mid i}(\sigma|S) \sum_{\sigma_{k\setminus k'}} \prod_{k \in \partial i \setminus k'} P_ki(\sigma|S)
\] (A8)

Exchanging again the sum and the product, but now on the last term, we get \( \sum_{\sigma_{k\setminus k'}} \left[ \prod_{k \in \partial i \setminus k'} P_ki(\sigma|S) \right] = 1 \), and using that \( r_i(S, S) = 0 \) and \( r_i(S, I) = \beta \) we obtain equation (11):

\[
\frac{dp_i}{dt} = -\mu p_i + \beta(1 - p_i) \sum_k p_{ki}
\] (A9)

Following the same steps is easy to derive the equation (12) for conditional probabilities.

**Appendix B: Average case equations for Erdos-Renyi graphs**

In this appendix we will show how to perform the closure of average-case equations for Erdos-Renyi graphs (sub-section V.B). Let’s start by explicitly writing the derivatives with respect to \( \kappa \):

\[
\frac{\partial \bar{p}}{\partial \kappa} = \frac{\partial}{\partial \kappa} \left( \sum_{\gamma} P(\gamma)p^\gamma \right) = \frac{\partial}{\partial \kappa} \left( \sum_{\gamma} \frac{e^{-\kappa \gamma + 1}}{(\gamma + 1)!}p^\gamma \right)
\] (B1)

\[
\frac{\partial \bar{\bar{p}}}{\partial \kappa} = \frac{\partial}{\partial \kappa} \left( \sum_{\gamma} g_{\text{link}}(\gamma)p^\gamma_{\gamma} \right) = \frac{\partial}{\partial \kappa} \left( \sum_{\gamma} \frac{e^{-\kappa \gamma}}{\gamma!}p^\gamma_{\gamma} \right)
\] (B2)

Computation of (B1) and (B2) gives:

\[
\frac{\partial \bar{p}}{\partial \kappa} = -\sum_{\gamma} \frac{e^{-\kappa \gamma + 1}}{(\gamma + 1)!}p^\gamma + \sum_{\gamma} (\gamma + 1) \frac{e^{-\kappa \gamma}}{(\gamma + 1)!}p^\gamma = -\bar{p} + \frac{1}{\kappa} \sum_{\gamma} (\gamma + 1) P(\gamma)p^\gamma
\] (B3)

\[
\frac{\partial \bar{\bar{p}}}{\partial \kappa} = -\sum_{\gamma} \frac{e^{-\kappa \gamma}}{\gamma!}p^\gamma_{\gamma} + \sum_{\gamma} \frac{e^{-\kappa \gamma - 1}}{\gamma!}p^\gamma = -\bar{\bar{p}} + \frac{1}{\kappa} \sum_{\gamma} g_{\text{link}}(\gamma)p^\gamma_{\gamma}
\] (B4)

The sums in the right hand sides of (B3) and (B4) are also involved in equations (20) and (21). Then, remembering that \( \kappa = \sum_{\gamma} (\gamma + 1) P(\gamma) \), we can rewrite equation (20) as follows:

\[
\dot{\bar{p}} = -\mu \bar{p} + \beta \bar{\bar{p}} \sum_{\gamma} (\gamma + 1) P(\gamma)(1 - p^\gamma)
\]

\[
\dot{\bar{\bar{p}}} = -\mu \bar{\bar{p}} + \beta \bar{\bar{p}} \sum_{\gamma} (\gamma + 1) P(\gamma) - \beta \bar{p} \sum_{\gamma} (\gamma + 1) P(\gamma) p^\gamma
\]

\[
\dot{\bar{\bar{p}}} = -\mu \bar{\bar{p}} + \beta \kappa \bar{p} - \beta \bar{p} \bar{\bar{p}} \left( \bar{p} + \frac{\partial \bar{p}}{\partial \kappa} \right)
\] (B5)
which leads directly to equation (24). Equation (25) can be derived by an analogous procedure, using an equivalent expression for the mean connectivity: \( \kappa = \sum \gamma g_{\text{link}}(\gamma) \)

Now we just need closed expressions for the derivatives in (24) and (25). In order to do so, let’s compute the \( \kappa \)-derivative on both sides of ansatz (26) and (27). We get:

\[
\frac{\partial \tilde{p}}{\partial \kappa} = \frac{1}{2} \left[ 1 - \tanh^2 \left( \beta \kappa \chi(t) - \mu \right) \right] \beta \chi(t) \tag{B6}
\]

\[
\frac{\partial \tilde{p} \rightarrow}{\partial \kappa} = \frac{1}{2} \left[ 1 - \tanh^2 \left( \beta \kappa \epsilon(t) - \mu \right) \right] \beta \epsilon(t) \tag{B7}
\]

We can re-use equations (26) and (27) for eliminating \( \chi(t) \) and \( \epsilon(t) \) from (B6) and (B7), thus obtaining the following closed expressions for the derivatives:

\[
\frac{\partial \tilde{p}}{\partial \kappa} = \frac{1}{2\kappa} \left[ 1 - (2\tilde{p} - 1)^2 \right] \left[ \tanh^{-1}(2\tilde{p} - 1) + \mu \right] \tag{B8}
\]

\[
\frac{\partial \tilde{p} \rightarrow}{\partial \kappa} = \frac{1}{2\kappa} \left[ 1 - (2\tilde{p} \rightarrow - 1)^2 \right] \left[ \tanh^{-1}(2\tilde{p} \rightarrow - 1) + \mu \right] \tag{B9}
\]

This allows to numerically solve equations (24) and (25).

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