Anomalous contagion and renormalization in networks with nodal mobility

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received 3 April 2016; accepted in final form 8 July 2016
published online 1 August 2016

PACS 89.75.-k – Complex systems
PACS 89.75.Hc – Networks and genealogical trees
PACS 87.23.Ge – Dynamics of social systems

Abstract – A common occurrence in everyday human activity is where people join, leave and possibly rejoin clusters of other individuals —whether this be online (\textit{e.g.} social media communities) or in real space (\textit{e.g.} popular meeting places such as cafes). In the steady state, the resulting interaction network would appear static over time if the identities of the nodes are ignored. Here we show that even in this static steady-state limit, a non-zero nodal mobility leads to a diverse set of outbreak profiles that is dramatically different from known forms, and yet matches well with recent real-world social outbreaks. We show how this complication of nodal mobility can be renormalized away for a particular class of networks.

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Introduction. – Significant attention among physicists has turned to problems where the dynamics of a meme or virus, plus the network structure on which it is spreading, co-evolve on comparable timescales —whether online \cite{1-3} or in real space \cite{4-13}. Thus a traditional epidemiological description based on mass-action differential equations becomes inadequate. There is a rich variety of works \cite{8-25} that reflect the many possible choices for how a network can evolve dynamically, and hence be modeled to account for detailed real-world mobility patterns \cite{6,7,12,26,27}, behavioral effects \cite{28} and environmental factors \cite{5}. In the online world, there are multiple ways in which humans can cluster, including social media communities. Each opens a new pathway for informational contagion (\textit{e.g.} rumor, idea or plan). Even if the number of individuals actively online in each community is fairly constant, there will be significant churn as people drop in, drop out, and drop back into the conversations over the course of a day, weeks or months \cite{3,22,29-32}.

Here we examine the impact of this common dynamical feature of everyday human behavior whereby people join, leave and can rejoin clusters of other individuals $C_1, C_2, \ldots, C_M$, \textit{e.g.} by sporadically checking online posts for a particular social media community or re-visiting a particular cafe. The dynamical complication comes from the fact that a returning individual (\textit{i.e.} node) may find a cluster of individuals (nodes) in which membership has changed very little or a lot, depending on the migration of all the other individuals (nodes). Depending on who they then meet after they enter or re-enter the community, and when, the resulting evolution of any meme or virus at the population level may be very different.

Our model for this co-evolution is purposely very simple (see fig. 1) so that we can write down, and solve numerically, coupled differential equations that mirror the outcome of numerical simulations, as well as enabling some analytical analysis. Yet our simple setup turns out to generate highly anomalous infection profiles which capture the diversity of those observed in recent periods of civil unrest that were fueled by social media (fig. 4). While we do not pretend that our model provides a unique explanation of these real-world phenomena, it serves the purpose of providing a more unified view of such collective social activity. Specifically, our analysis shows that even though a network may appear static on average, an underlying nodal mobility can generate highly nonlinear behavior in an outbreak’s severity (\textit{i.e.} peak infection value $H$), time-to-peak (\textit{i.e.} time $T_m$ from beginning of outbreak to its peak), duration $T$, and area $A$ under the profile $I(t)$. We also provide a novel renormalization scheme that can significantly reduce the complexity of this class of dynamical network problem.
Network model. – We assume here for simplicity that the $M$ network clusters are internally fully connected (fig. 1(a)) and that for $M > 1$ the different clusters are interconnected in simple ways, e.g. parallel (fig. 1(c)) and series (fig. 1(d)) by means of sparse links. Given any particular network architecture containing clusters in parallel, series or a combination, our goal is to better understand the impact of allowing nodes to migrate into, out of, and between these clusters such that the overall network appears static on average and yet the identity of the nodes within the clusters can change over time. Consider first a network of $N$ total nodes in which there is a single cluster $C_1$ (fig. 1(a)). At any given timestep, a node from anywhere outside $C_1$ has a probability $p_1$ to join $C_1$, while a node inside $C_1$ has a probability $p_{l1}$ to leave $C_1$. The number of nodes $N_1(t)$ in $C_1$ follows $N_1 = -p_1 N_1 + p_l (N - N_1)$. For the steady-state situation where $N_1 = 0$, the mean cluster size is constant and so the network appears structurally static on average, ignoring individual nodal identity. This mean size $\langle N_1 \rangle = N p_l (p_1 + p_{l1})^{-1} \equiv N \gamma_s$ and the sum of the mean number of nodes joining and leaving is $\mu = (N - \langle N_1 \rangle) p_1 + \langle N_1 \rangle p_l \equiv N \gamma_m$. Hence $\gamma_s = p_l (p_2 + p_{l2})^{-1}$ characterizes the mean size of $C_1$ and $\gamma_m = 2 p_1 p_l (p_1 + p_{l1})^{-1}$ characterizes the nodal mobility through $C_1$. At any timestep, an infected agent within $C_1$ transmits a meme or virus to any susceptible within $C_1$ with probability $q$, (fig. 1(b)). Since $C_1$ is the only fully connected cluster, we will assume that transmission from infected nodes outside $C_1$ is negligible by comparison.

Since recovery is individual based, infected nodes inside and outside $C_1$ have probability $q_i$ to become immune (for SIR) or susceptible again (for SIS). The infection rate $\lambda = q_i/q_s$ is the usual ratio of the infection probability to the recovery probability. Since the $S\rightarrow I$ process only occurs inside the cluster, we use $S(t), I(t), R(t)$ for the number of susceptible, infected, and recovered nodes in the whole system, and $S_1(t), I_1(t)$, and $R_1(t)$ for the corresponding numbers in the cluster. The six equations that describe the dynamics of an SIR process in this single dynamical cluster situation, are

$$\dot{S}_1(t) = -q_i S_1 I_1 + q_i I_1 - p_1 (S_1 - S_1 I_1),$$
$$\dot{I}_1(t) = q_i S_1 I_1 - p_1 (I_1 + q_i S_1 I_1 - q_i I_1) + (1 - q_i) p_1 (I - I_1),$$
$$\dot{R}_1(t) = q_i I_1 - p_1 (R_1 + q_i I_1) + p_1 ((R - R_1) + q_i (I - I_1)),$$
$$\dot{S}(t) = -q_i S_1 I_1,$$
$$\dot{I}(t) = q_i S_1 I_1 - q_i I,$$
$$\dot{R}(t) = q_i I.$$

For an SIS process, there is no immunity for recovered nodes. Hence there are only four dynamical equations:

$$\dot{S}_1(t) = -q_i S_1 I_1 + q_i I_1 - p_1 (S_1 - S_1 I_1),$$
$$\dot{I}_1(t) = q_i S_1 I_1 - p_1 (I_1 + q_i S_1 I_1 - q_i I_1) + p_1 (1 - q_i) (I - I_1),$$
$$\dot{S}(t) = -q_i S_1 I_1 + q_i I,$$
$$\dot{I}(t) = q_i S_1 I_1 - q_i I.$$

In the simulations, all nodes are initially susceptible and we allow the system to run until the cluster size reaches its steady-state size $N\gamma_s$. We then randomly pick a node in $C_1$ and make it infected. In every subsequent timestep, all the nodes first carry out the SIR (or SIS) process followed by the joining or leaving of $C_1$. We choose $N = 1000$. Figure 2(a) shows the trajectory of $S$ and $I$ values in the $M = 1$ cluster model with SIR, in $S$-$I$ space. The trajectory starts from the lower right-hand corner, as initially we have $S/N \sim 1$ and $I/N \sim 0$. The results are in sharp contrast with the standard SIR model in a well-mixed population in which once the infection rate $\lambda$ and initial number of infecteds $I(0)$ are given, the trajectory is fixed [33]. For the standard SIR in a well-mixed population, if $\lambda$ and $I(0)$ are given, then there will only be one trajectory in the $S$-$I$ space. The number of recovered nodes $R$ at the end of the outbreak reflects the extent of the infection. We stress that this is real oscillatory behavior, not simply fluctuations. These oscillations (or more generally, resurgent behavior) also appear in results obtained from integrating the set of equations (eq. (1)), although the resulting curve is smoother as an average over many runs is implicitly implied by the equations. In particular, the resurgence arises from the occasional supply of susceptible and infected when nodes join the cluster. For systems larger than 20000 nodes, the oscillations
We find that the effect of nodal mobility and cluster size on the steady state of the infection is highly nonlinear.

While it is known that models with heterogeneity in connectivity or nodal type can produce anomalous infection characteristics as compared to the usual well-mixed SIR model, our model shows this can arise in a network that appears static on average and in which the time-averaged properties of each node are the same, i.e. anomalous infection profiles arise even though each node spends the same average time in cluster $C_1$ and has the same average number of links over time. Figure 2(c) examines the effect of the nodal mobility and infection rate on the outbreak’s severity. We find that for small infection rate $\lambda < 0.15$, there is a monotonic nonlinear decrease of the outbreak severity with increasing nodal mobility $\gamma_m$. This might be expected since spending less time in the cluster exposes an individual (i.e. mobile node) to less risk of infection. However, one could imagine a competing mechanism whereby increased mobility helps refuel the number of infected in a cluster. As $\lambda$ increases, the interplay of these two yields a critical value of $\lambda \approx 0.15$. A maximal severity now emerges at finite $\gamma_m$ obeying the approximate relationship $\gamma_m \sim e^{0.005 q_i}$. For a given infection probability $q_i$, the critical value of $\gamma_m$ separates a low-$\gamma_m$ phase in which increasing nodal mobility yields a decrease in outbreak severity, and a high-$\gamma_m$ phase in which increasing $\gamma_m$ yields an increase in severity.

**Two-cluster version and social outbreaks.** – Clusters in parallel (fig. 1(c)) or series (fig. 1(d)) mimic individuals who access one type of space such as a Facebook community, either at the same time as they check another (parallel case) or before they check another (series case). Figure 3 illustrates the rich infection profile behavior $I(t)$ that emerges for parallel (left column) and series (right column) clusters with person-to-person contagion. For the parallel case, we make the simple choice that an agent joins clusters $C_1$ or $C_2$ with probabilities $\omega_p$ and $1 - \omega_p$, respectively. Hence in the steady state, $\langle N_i \rangle = \gamma_s N$, for $i = 1, 2$, with $\gamma_{s,1} = \frac{p_i p_j}{p_i + p_j} \omega_p$, and $\gamma_{s,2} = \frac{p_j}{p_i + p_j} (1 - \omega_p)$. The $M = 1$ case is recovered as $\omega_p \rightarrow 1$ or $\omega_p \rightarrow 0$. Figures 3(b) and (c) show that the infection peak height $H$ decreases significantly as $\omega_p \rightarrow 1/2$, while fig. 3(a) shows a local maximum at $\omega_p = 1/2$. These behaviors are favored by the average size of each cluster becoming similar as $\omega_p \rightarrow 1/2$.

For the series case, we make the simple choice that an agent in $C_1$ joins cluster $C_2$ with probability $\omega_s$ and so

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**Fig. 2:** (Color online) (a) Trajectories of evolution of the system in the $S-I$ space for three different sets of parameters. The rough and smooth curves are obtained by numerical simulations and integrating the set of equations, respectively. The parameters are: $\gamma_m = 0.009$, $\lambda = 0.1$, and $q_i = 0.005$ (red curves); $\gamma_m = 0.018$, $\lambda = 0.1$, and $q_i = 0.001$ (green curves); $\gamma_m = 0.0018$, $\lambda = 0.022$, and $q_i = 0.002$ (blue curves). The insets show the time dependent of $I_m(t)$ for each of the cases. (b) SIS (Susceptible-Infected-Susceptible) for one-cluster version. Vertical scale is $I(\infty)/N$, the normalized fraction of infected nodes in the long-time limit, as a function of $\gamma_m$. $N = 1000$, $q_i = 0.0005$, $q_r = 0.015$. $p_i + p_j = 1$ for simplicity. The inset shows $I(\infty)/N$ as a function of $\gamma_s$. Lines are from integrating the coupled differential equations, symbols are simulation results. (c) Nonlinearity of SIR outbreak severity ($I(t)$ peak height $H$ divided by the constant $N$ which is total number of network nodes) as a function of nodal mobility $\gamma_m$ and $q_i$, for different values of the ratio $\lambda = q_i/q_r$ for one-cluster version (fig. 1(a)).

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Fig. 3: (Color online) Infection profile $I(t)$ vs. time (vertical axis) for different $M = 2$ cluster geometries and model probabilities (horizontal axis). (a)–(c) Clusters in parallel (fig. 1(c)). Profiles calculated by numerical integration of differential equations (see SM) for three $(d)$–$(f)$ Clusters in series (fig. 1(d)). Profiles calculated by probabilities (horizontal axis). (a)–(c) Clusters in parallel (fig. 1(c)).

For $M > 2$. An agent in the final cluster $C_M$ leaves it with probability $p_l$. Hence in the steady state for $M = 2$, the differential equations for the population in each cluster become:

$$\frac{dN_1}{dt} = -\omega_s N_1 + p_j (N - N_1 - N_2),$$

$$\frac{dN_2}{dt} = -p_l N_2 + \omega_s N_1,$$

In the long-time limit, the mean number of nodes in $C_1$ and $C_2$, respectively, can be written in terms of the mean size parameter for the single cluster case, $\gamma_s$:

$$\langle N_1 \rangle = N \gamma_s \left( \frac{\omega_s}{p_l} + \frac{p_j}{p_l + p_j} \right)^{-1},$$

$$\langle N_2 \rangle = N \gamma_s \left( 1 + \frac{p_j}{\omega_s p_l + p_l} \right)^{-1}.$$

For $\omega_s < p_l$, $\langle N_1 \rangle \rightarrow N/(1 + \kappa)$, where $\kappa = \omega_s/p_j$, while $\langle N_2 \rangle \rightarrow 0$. By contrast for $\omega_s \gg p_l$, $\langle N_1 \rangle \rightarrow 0$ while for $C_2$ we recover the equilibrium population for the single cluster version (fig. 1(a)). The asymmetry in figs. 3(d)–(f) for $M = 2$ clusters in series is strikingly different from the symmetry shown for the parallel case (figs. 3(a)–(c)). This asymmetry has its roots in the breaking of symmetry in time (i.e. a node passes through $C_1$ before $C_2$). In addition, figs. 3(d)–(f) also show that as $\omega_s$ grows, the infection profile gradually becomes less dependent on it.

Indeed, when $\omega_s$ grows much larger than the probability of leaving $C_2$, $p_l$, the profiles become identical to the $M = 1$ case and $\omega_s$ becomes irrelevant. Thus, the infection profiles for the series case experience their largest variation for small $\omega_s$, with infection peaks that are significantly higher than for larger $\omega_s$. This is because at low $\omega_s$, $C_1$ has many nodes on average and these nodes are more likely to get infected and hence infect others. As $\omega_s$ grows much greater than $p_l$, the size of $C_2$ approaches the $M = 1$ case while the size of $C_1$ falls to zero. As a result, the infection profiles become identical to the $M = 1$ case.

Next, we compare the variability and saturation effect in the time-to-peak $T_m$ for one- and two-cluster versions. So far we have looked at epidemics that require at least one infected node within the cluster so that they could arise. We call this a person-to-person mechanism. An alternative case that we consider now is a broadcast mechanism, in which the nodes residing on one of the clusters experience a constant probability of getting infected regardless of the number of infected nodes present in the cluster. For the one cluster version, we will use the standard person-to-person infection mechanism. For the two-cluster version, we will choose the first cluster to have person-to-person infection while the second is broadcast, e.g. the first cluster mimics individuals in an online chatroom community while the second mimics individuals listening to the same radio broadcast. Two-cluster combinations, with this choice of infection mechanism, can produce a larger ratio $T_m/T$ when compared with the one-cluster version, as illustrated in fig. 4(a). Specifically $T_m/T > 0.5$ as observed in the empirical data (fig. 4(b)). Interestingly, the two-cluster series combination in fig. 4(a) yields a near constant ratio $T_m/T$ for small values of mobility $\gamma_m$ but $T_m$ saturates as $T$ increases for larger $\gamma_m$ – by contrast, the one-cluster version shows the opposite trend. This calculation for the parallel model reveals an analogous trend to the one-cluster version with minimal variations, hence it is not shown in the figure.

The on-street civil unrest data (colored dots) in fig. 4(b) come from a unique multi-year, national research project involving exhaustive protest event analysis by subject matter experts (SMEs) across Latin America (see refs. [35,36]). The start and end of each outbreak is identified using the analysis of ref. [37] and cross-checked manually. Figure 4(b) shows how the time-to-peak ($T_m$) and duration ($T$) of civil unrest outbreaks (color dots) relates to those generated from our model. The single cluster model captures outbreaks where $T_m < T/2$ (see middle and bottom simulation curve in fig. 4(b)) while $M = 2$ clusters in series extends the model’s descriptive range to $T_m > T/2$ in agreement with the data (fig. 4(b) main panel). This is consistent with individuals in the real world, as in the model, sporadically joining and leaving online and/or offline communities in which the idea of creating on-street protests is beginning to circulate. Hence the model’s outbreak profile and the actual on-street protest outbreak profile should look similar, as they do.
Renormalization and circuit equivalence. – The general case of \( M > 2 \) clusters allows for an interesting connection between the nodal migration dynamics and electric circuits (fig. 1(e)) and a novel renormalization. We use \( K \) as the cluster label, \( p_k \) as the probability to leave cluster \( k \), and \( n_k \) as the number of nodes in cluster \( k \). We can associate an effective resistance \( R_k \equiv 1/p_k \), potential difference \( U_k \equiv n_k \) and current \( i_k \equiv \Delta n_k = U_k/R_k = n_k p_k \). This equivalence allows us to then generalize our model to \( M \) clusters connected either in series or in parallel and hence quantify its dynamics. We have established this mapping exactly for \( M > 2 \) clusters that are either all in series or in parallel. As an illustration in the steady state, the number of nodes in each of \( M \) clusters connected either in series or in parallel is as follows:

\[
\begin{align*}
    n_{0}^{(s)} &= \frac{N}{p_0} \left( \sum_{i=0}^{M-1} \frac{1}{p_i} \right)^{-1}, \\
    n_{k}^{(s)} &= \frac{N}{p_k} \left( \sum_{i=0}^{M-1} \frac{1}{p_i} \right)^{-1}, \\
    n_{0}^{(p)} &= \frac{N}{p_0} \left( \frac{1}{p_0} + \sum_{i=1}^{M} \frac{p_{0,i}}{p_{0,i}} \right)^{-1}, \\
    n_{k}^{(p)} &= \frac{N p_{0,k}}{p_k} \left( \frac{1}{p_0} + \sum_{i=1}^{M} \frac{p_{0,i}}{p_{0,i}} \right)^{-1},
\end{align*}
\]

where \( k = 0 \) represents the nodes outside the fully connected set of clusters; \( N \) is the total number of nodes; \( p_{0,j} \) is the probability of moving from cluster 0 to cluster \( j \) and superscripts \( s \) and \( p \) denote series and parallel cases, respectively. For the series case, we can then regard the first \( (M-1) \) clusters as a renormalized super-cluster \( 1' \) and replace the last cluster by cluster \( 2' \) with the following steady-state populations:

\[
\begin{align*}
    \langle N_1' \rangle &= N \gamma_n \left( \omega_n' p_i + \frac{p_j}{\omega_n' p_i + p_j} \right)^{-1}, \\
    \langle N_2' \rangle &= N \gamma_n \left( 1 + \frac{p_i}{\omega_n' p_i + p_j} \right)^{-1},
\end{align*}
\]

where \( \omega_n' = (\sum_{k=1}^{M-1} \omega_k^{-1})^{-1} \) is the effective probability of nodes from cluster \( 1' \) migrating to cluster \( 2' \). \( \omega_k \) is the migration probability from cluster \( k \) to adjacent node \( k+1 \) in series. Similarly, for the parallel case we can implement an analogous renormalization so that the size of the super-cluster \( 1' \) and cluster \( 2' \) can be written as

\[
\begin{align*}
    \langle N_1' \rangle &= N \gamma_n \omega_n', \\
    \langle N_2' \rangle &= N \gamma_n (1 - \omega_n'),
\end{align*}
\]

where \( \omega_n' = \sum_{i=1}^{M-1} \omega_i \) is the effective probability of joining the super-cluster while here the product \( p_j \omega_n \) is the probability to join cluster \( i \). With this renormalization, effective two-cluster differential equations can then be written down and solved for the general \( M \) case.

Conclusions. – In summary we have shown that nodal migration through a network generates highly complex outbreak profiles, even though the network may appear static on average. The introduction of this dynamical feature yields infection patterns that mimic empirical data, specifically outbreaks of civil unrest. These patterns lie beyond the scope of traditional epidemiological models. In addition, we find significant differences in the infection profiles for broadcast transmission within a cluster as compared to person-to-person. This suggests distinct containment policies should be explored for outbreaks whose root cause is infected transient individuals (e.g. hospital patients or airline travelers) as opposed to infected transient places (e.g. the hospital or airport itself). We have also indicated how the complex throughput of nodes can be renormalized exactly for a particular class of dynamical network.

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We are grateful to CHAO-MING SONG and STEFAN WUCHTY for detailed discussions. NFJ gratefully acknowledges support from National Science Foundation (NSF) grant CNS1500250 and Air Force (AFOSR) grant 16RT0367.

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