Disease-induced resource constraints can trigger explosive epidemics

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Abstract

Advances in mathematical epidemiology and network science have led to a better understanding of the risks posed by epidemic spreading and informed strategies to contain disease spread through prevention and treatment. However, a challenge that has been overlooked is that, as a disease becomes more prevalent, it can limit the availability of the capital needed to effectively treat those who have fallen ill. Here we use a simple mathematical model to gain insight into the dynamics of an epidemic when the recovery of sick individuals depends on the availability of healing resources that are generated by the healthy population. We find that epidemics spiral out of control into "explosive" spread if the cost of recovery is above a critical cost. This can occur even when an epidemic would not be predicted by a standard model that does not consider the reduction in resource generation as individuals fall ill. Through simulations and analytical solutions we show that the onset of explosive epidemics is very sudden, exhibiting a discontinuous transition under very general assumptions. We also determine analytically the critical cost and the size of the explosive jump in infection levels in terms of the parameters that characterize the spreading process. Our model and results apply beyond epidemics to other contagion dynamics that self-induce constraints on recovery, thereby amplifying the spreading process.

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2

I. INTRODUCTION

The global mobility of individuals that sustains modern economic activity has increased the risk of pandemics [1, 2]. For example, approximately 3.1 billion passengers travel by air every year, accelerating disease spread and making spreading patterns more complex [3, 4]. The biggest pandemic on record, the 1918 Spanish influenza, was responsible for the death of 50 million people at a time when global mobility was slower and much less. Thus, while the containment and mitigation of global disease spread in our time has become harder due to high interconnectedness, it could save millions of lives, making it one of the most pressing 21st-century challenges. Important progress in understanding epidemic spreading has been made using mathematical models that capture the underlying processes and their dependence on infection and recovery rates [5–7]. These studies have focused mainly on the interplay between the dynamics and the structure of interactions [4, 8–12], and on identifying the main spreaders [3, 13, 14]. Progress has also been made in developing strategies to mitigate epidemic risks, for example through vaccination [9, 15–20]. Many studies address the inherent limitations in the resources available to treat or prevent diseases, for instance by finding the optimal way to allocate a limited pool of immunization doses and antivirals [21]. This work assumes that the resources available to treat and prevent disease are independent of the evolution of the epidemic. However, the health burden a disease places on a population can limit the production of these resources [22]. Here we investigate a simple model that includes such disease-induced resource constraints and find that they can amplify epidemics. In the most extreme cases the disease will rapidly invade the whole population even if healing resources could have effectively contained it.

A healthy and productive population is needed to enable the recovery of those that become ill: Healthy individuals are both the human resources needed to provide health services and pharmaceuticals, and contributors to the healthcare budget through taxes and insurance premiums. These mechanisms operate on shorter and longer timescales but the availability of resources is always most relevant for diseases where the cost of treatment is high. This can apply to diseases that are endemic in a population and have depressed the economic productivity and growth of a region over a sustained period, leading to an underfunded health care system that cannot provide the necessary level of treatment. For example, it has been estimated that countries where Malaria was widespread in 1965 had a 1.3% lower

annual growth rate than other countries between 1965 and 1990 [23, 24]. Similarly, AIDS could lead to the collapse of many African economies within a few years [25]. On the other hand, resource constraints also apply to sudden outbreaks that evolve too fast for the hospital and drug production systems to adapt the available resources to supply sufficient treatment in the necessary time frame. A timely example are highly virulent emergent pathogens, such as SARS and H1N1 influenza, which are widely feared because of their potential to generate the next great pandemic [26, 27]. An even more recent example is the Ebola epidemic unfolding in West Africa, where one of the main initial challenges was the high infection rate in the already limited population of nurses and doctors, due partly to understaffing and suboptimal protective practices at treatment centers [28, 29].

To investigate the effect of disease-induced resource constraints on epidemic spreading we consider the simplest epidemiological model that captures longer time scales, the *Susceptible-Infected-Susceptible* (SIS) model. This model applies directly to infections that do not confer immunity, such as many bacterial infections, but also to the general case where a population is exposed to a number of diseases, and individuals that have been infected by one disease may easily become infected by another. It is also a good approximation for cases where immunity is conferred but it wanes rapidly relative to the infection dynamics. The specific model we use applies to cases where no external resources can enter the system – either the disease is so widespread that there are no unaffected regions from which resources can be transferred, or transfer cannot occur for logistical or political reasons. We emphasize that our goal is not to capture the detailed dynamics of specific diseases, but rather to understand what type of behavior can emerge due to disease-induced resource constraints, and to characterize analytically how this behavior depends on the basic parameters of a contagion model. We also seek to determine the generality of our findings, and will discuss some cases where simulations or analytic arguments indicate that qualitatively similar behavior arises independent of model assumptions.

A. Epidemic model with endogenously generated healing resources

In the SIS formalism individuals are either in a susceptible state or in an infected state. We will denote by $s(t)$ and $i(t)$ the fraction of susceptible and infected individuals, respectively. Susceptible individuals are healthy but become infected at a characteristic infection

rate p when interacting with infected individuals. Infected individuals recover and become susceptible again at a recovery rate q. In this model individuals recover independently of available resources.

To model a recovery process that is dependent on healing resources generated by the healthy population, we introduce our budget-constrained Susceptible-Infected-Susceptible (bSIS) model illustrated in Fig. 1. This model modifies the SIS dynamic by (1) introducing a budget b that increases by one unit with the number of healthy individuals per unit time and (2) constraining recovery, at least partially, if the necessary budget to cover costs of healing is not available. In other words, there is a background recovery process that is independent of the budget available, and this process occurs at a rate q_0. However, to reach the full potential for recovery, resources must be invested. Specifically, a cost c is incurred for each individual that recovers through treatment. (Note that this model captures the most general case, defining costs relative to the resource unit generated per unit time.) Clearly the rate of resource mediated recovery will depend on the total budget b available. If there is no budget, there will be no resource mediated recovery. On the other hand, with a positive budget some recovery is possible, and the rate of recovery increases until the maximum resource mediated recovery rate q_b is reached. To express this dependence we introduce the budget function $f(b)$ which satisfies $f(b) \equiv 0$ for $b \leq 0$ and $0 < f(b) \leq 1$ for $b > 0$. Thus, the resource mediated recovery rate, given budget b, is just $q_b f(b)$ as shown in Fig. 1.

The simplest budget function fulfilling the required conditions is the Heaviside step function $\Theta(b)$, which is one for $b > 0$ and zero otherwise. In fact, for the sake of analytical tractability, this is the function we will consider for most of our analysis. However, we will show in the *Generalized Behavior* section that the key results we derive using this specific budget function hold under any budget function that satisfies the general conditions defined above.

B. Interaction structure and the mean-field approximation

To define the spreading dynamics we need to specify the interaction process between infected and susceptible individuals. One common starting point, which yields powerful insight and is analytically tractable, is the mean-field approximation. Topologically, the mean-field limit corresponds to an uncorrelated random graph, where every individual interacts with

5

k randomly selected others. With this assumption the dynamics of the fraction of infected individuals are given by

$$\frac{di(t)}{dt} = kpi(t)s(t) - [q_0 + q_b f(b)]i(t), \tag{1}$$

where $s(t) = 1 - i(t)$, so that the rate equation for the fraction of susceptibles is analogous. The first contribution on the right-hand side accounts for the infection of susceptible individuals that are in contact with infected ones, assuming an infection rate of p. The last term accounts for both the background and the budget dependent recovery processes.

To fully describe the evolution of the mean-field bSIS system we couple Eq. (1) with the equation for the rate of change in the budget $b(t)$:

$$\frac{db(t)}{dt} = s(t) - cq_b f(b)i(t). \tag{2}$$

where the first term accounts for the budget produced by healthy individuals, and the second term for the reduction in budget due to successful application of healing resources. Note that setting $f(b) \equiv 1$ for all b we recover the pure SIS system with recovery rate $q = q_0 + q_b$ (in this case Eq. (2) is redundant).

One of the main insights obtained from the SIS model without budget constraints, is the existence of a transition from a *healthy* regime where the fraction of infected individuals vanishes in the thermodynamic limit (infinite system size), to an *epidemic* one characterized by a non-negligible fraction of infected individuals [7]. The details of the transition depend on the interaction structure between individuals [30, 31]. In the mean-field system the effective base reproduction number $\tau = kp/q$, which is just the number of secondary infections generated by one infected individual in a population of susceptible individuals, fully characterizes the onset of the SIS epidemic transition. Specifically, the system is in the epidemic regime when τ is greater than the critical value $\tau^* = 1$ [32], and the transition to the epidemic regime is continuous. Furthermore, the level of infection in the epidemic regime increases with the base reproduction number. Importantly, the steady state infection levels in the population are independent of the initial condition in such a model (as long as the initial infection level is greater than zero).

The introduction of a budget-limited recovery process transforms these dynamics by modifying the steady state infection level and introducing a dependence on initial conditions. Given the carefully chosen simplifying assumptions of this model, we are able to solve the

6

bSIS mean-field system described by Eqs. (1) and (2) analytically to understand the dynamics under a limited budget. This simple approach is especially powerful since we can also show that the same behavior applies when we consider more realistic interaction structures between individuals. Specifically, in real systems disease transmission occurs through non-random interactions, in contrast to the assumptions of the mean-field approximation. The interactions can be modeled as networks generated through the interplay between multiple social mechanisms [33, 34]. These generating mechanisms together lead to complex structural features that are not generically found in random networks,most importantly: high clustering coefficients [35], community structure [36, 37], and the small-world property [33]. Because the dynamics of disease spread can be highly sensitive to the interaction structure, we study the limited resource case on a real social network alongside the mean-field approximation. Specifically, we consider a school friendship network constructed from an extensive survey shown in Fig. 2a (see *Materials and Methods* for details), which exhibits the features characteristic of real social networks: clustering, community structure and the small-world property.

II.   RESULTS

A.   The emergence of explosive epidemics

We first investigate the behavior of the bSIS model on the friendship network, in a parameter regime where we would observe an epidemic in the SIS model ($\tau > \tau^*$). To build intuition we will initially consider the simplest case, where *all* healing requires a budget contribution ($q_0 = 0$), and in addition we are assuming a Heaviside step function for the budget. Thus, unless otherwise specified, $q = q_b$ in the results that follow. However, in the *Generalized behavior* section we will show that the key characteristics in the behavior also hold qualitatively when there is background healing and for more general functions.

In the early stages of the epidemic simulation we observe exactly the SIS behavior, as seen in Fig. 2b. We will refer to this, budget unrestricted infection incidence level, as the *low* epidemic. However, after a critical time $t = t^*$ the epidemic spreading rapidly escalates— a situation that we describe as an "explosive epidemic". We will refer to this increased incidence level as the *high* epidemic. We see in Fig. 2b, an explosive epidemic where the

7

entire population is infected in the steady state ($i(\infty) = 1$).

What leads to this drastic alteration of the SIS steady state infection level when we consider disease-induced budget constraints? The explosive epidemics arise when the healing budget is depleted and the normal recovery rate cannot be sustained. Depending on the healing cost c, one of the three qualitatively different behaviors of the budget in time (see Fig. 3), will be instantiated. When healing costs are small, the budget systematically increases and the system evolves towards an infinite budget. On the other hand, with large enough healing costs, the budget is used up at a finite time $t = t^*$. After this time, any contribution to the budget by healthy individuals is immediately spent on the recovery of only a small fraction of all the infected individuals. Thus the epidemic spread outcompetes recovery, further decreasing the number of healthy individuals who can contribute to the budget. Eventually this process leads to a fully infected population. In between these two regimes, there is a critical cost ($c = c^*$) at which the budget saturates at a non-zero value. Here the infection level evolves as it would without a budget constraint, until it eventually exhausts the budget. To approximate all three budget behaviors as observed in Fig. 3a we can simply integrate the mean-field system without a budget constraint ($f(b) \equiv 1$) (see *Materials and Methods* for the analytic solution).

The crucial point is that we can find a critical cost c^* above which positive contributions by healthy individuals to the healing budget are insufficient to meet the costs generated by the recovering patients (i.e. $s(t) < cqi(t)$). Thus, according to Eq. (2), the budget will start decreasing until it is depleted in finite time and we are in the second case described above, where infection takes over the population. Clearly, for a fixed cost and recovery rate, the resource demands for recovery are larger when there are more infected individuals. This means that the critical cost is determined by the maximum infection level that the system will reach before the budget starts to decrease, $\max_t[i_{\not{b}}(t)]$, where $i_{\not{b}}(t)$ denotes the infection level without a budget restriction. Putting all of this together, in the mean-field case we can find c^* by solving for c at the stationary state of the budget, that is we set $db(t)/dt = 0$ in Eq. (2) with the constraint that the steady-state infection level must be $\max_t[i_{\not{b}}(t)]$. This gives

$$c^* = [kp \max_t [i_{\not{b}}(t)]]^{-1}. \tag{3}$$

In the case we consider in Fig. 2 the effective disease reproduction number is above the critical level (i.e. $\tau > 1$), and $0 < i(0) < 1 - \tau^{-1}$, which means that the pure SIS dynamic

reaches the maximum infection level at the epidemic equilibrium $i(\infty) = 1 - \tau^{-1}$. This gives the special case of Eq. (3)

$$c^* = (kp - q)^{-1} . \tag{4}$$

This case applies when $\tau > 1$, as long as the initial infection level is not larger than the SIS epidemic equilibrium (i.e. $0 < i(0) \leq 1 - \tau^{-1}$). If the initial level of infection $i(0)$ is larger than the pure SIS equilibrium level then $\max_t[i_b(t)] = i(0)$, whatever the value of τ. We can see this by noting that infection cannot increase above $i(0)$ if $b > 0$, since the pure SIS dynamics apply and in the SIS model infection monotonically converges to the equilibrium. Thus Eq. (3) implies that in this case the critical cost depends on the initial condition. In fact, at the critical cost $c = c^*$ the recovery demands must immediately match the generated resources. Since the initial budget is $b(0)$, the infection level will oscillate around the initial infection level $i(0)$ as the budget continuously switches on and off. This indicates that it is critical to treat diseases before the fraction of infected people escalates above the epidemic level since otherwise the epidemic will become explosive more easily (i.e. c^* will be smaller).

Interestingly, this last case implies that even for $\tau \leq 1$, where no epidemic phase is predicted in a pure SIS model, in the bSIS model we find a finite critical cost above which infections spread without efficient healing (dependent on the initial condition $i(0) > 0$). Furthermore, the outcome of a disease outbreak in the SIS model can be fully characterized by the effective base reproduction number $\tau = kp/q$, but in the bSIS model two parameters are needed: kp (the effective contact rate) and q (the recovery rate). These represent the time scales of infectious contact and recovery, respectively. While the dynamics of infection are still described by τ, the budget evolution depends on the competition between the time scales of recovery and budget generation, thereby requiring one additional parameter.

Can we determine the critical time at which the budget is exhausted, given the cost of treatment? We would expect that the larger the healing costs, the faster the budget is exhausted. We do in fact observe in our simulations that the critical time t^* scales with the healing cost as $t^* \sim (c - c^*)^{-1}$ (see Fig. 3b). Using the mean-field model we can calculate t^* by finding the point at which the budget is exhausted in the case $c > c^*$ (for details see *Materials and Methods*). For the case $\tau > 1$ we can use Eq. (8) in *Materials and Methods* to show that $t^* \sim (c - c^*)^{-1}$ as found in the friendship network. If $\tau \leq 1$, one obtains $t^* = 0$ as a consequence of the initial condition $b(0) = 0$, which implies that the treatment is constrained by the available budget from the beginning. The contributions of healthy

9

individuals to the budget cannot cover the treatment costs.

B. Explosive epidemic transitions are discontinuous

The dynamic change at t^*, and the consequent drastic increase in the fraction of infected individuals, is a continuous process in time (see Fig. 2b). However, the transition between the regimes is discontinuous in the model parameters (the healing costs as well as the recovery and infection rates). For example, as illustrated in Fig. 4, the transition is always discontinuous in the effective base reproduction number τ. The transition is also discontinuous in the sense that there is a jump, Δi_∞, in the infection level (see Fig. 3b). We discuss below how the jump size scales with the model parameters. The discontinuity of this jump in the final infected fraction $i(\infty)$ has important implications for the resilience of the health system and the control of disease, since it implies that a minute change in the properties of the disease or average contacts of an individual can abruptly shift the infection levels from a low equilibrium to one that is higher and harder to control.

C. Explosive epidemics arise across network structures

It is surprising that we can capture most of the dynamics of the epidemic on a real social network using only mean-field calculations. In particular, the same critical exponents are found for the scaling of both the fraction of infected individuals and the critical time with the distance from the critical cost. This leads us to ask whether the results we have found above are independent of network structure. We hypothesize that there is a resemblance between the dynamics in the random network of the mean-field approximation and the real friendship network, because the long-range interactions present in both networks dominate the dynamics. To test this hypothesis we study the bSIS model on a square lattice, where there are no long-range connections, and we would therefore expect to find different scaling exponents if our hypothesis is true. As shown in Fig. 4b we again find that the transition between the low and high epidemic regimes is discontinuous. However, in the epidemic regime $i(\infty) \sim (\tau - \tau^*)^\beta$ with $\beta \approx 0.59$. This value is instead consistent with the order parameter exponent $\beta = 0.586 \pm 0.014$ reported for the classical SIS on the square lattice [38]. However, we find that the critical time scales with the distance from the critical cost according

to $t^* \sim (c - c^*)^b$, with $b = -0.98 \pm 0.03$, which is numerically consistent with the one found in the mean-field limit (see *Supplementary Information*). This suggests that the effects of the cost of recovery on the system dynamics are similar across network topologies.

D. Generalized Behavior

The existence of an "explosive epidemic" that is discontinuous in the model parameters and the dependence of the system behavior on the initial conditions also holds when we allow for a baseline recovery rate $q_0 > 0$, as can be seen in the specific case shown in Fig. 5. The generality of our results can be easily appreciated if we define c^* as the cost above which the dynamics will converge to a stable equilibrium infection level that is higher than the equilibrium infection level in the model without resource constraints, $i_{\cancel{b}}(\infty) = 1 - \tau^{-1}$, where $q = q_0 + q_b$. We note that in this model infection will always die out, independent of the budget, if $\tau_{q_0} = kp/q_0 < 1$. Thus in what follows we only consider the case where $\tau_{q_0} \geq 1$. Solving for the fixed point of the budget by setting $db(t)/dt = 0$ in Eq. (2) and substituting into Eq. (1) we can express the full infection dynamics using

$$\frac{di(t)}{dt} = kpi(t)[1 - i(t)] - c^{-1}[1 - i(t)] - q_0 i(t). \tag{5}$$

As c approaches infinity the resource mediated recovery is no longer possible and this system becomes the SIS model with recovery rate q_0, which has a stable equilibrium that is greater than $1 - \tau^{-1}$, namely $1 - \tau_{q_0}^{-1}$. However, we can also show that a finite c^* exists. Solving for the fixed points of Eq. (5) we find that there are two fixed points $i(\infty)^+ \geq i(\infty)^-$ (given by Eq. (9) in *Materials and Methods*). We note that for any finite cost c, $i(\infty)^+$ is smaller than the equilibrium of the SIS model with recovery rate q_0. However, we can define a c^* that guarantees that $i(\infty)^+ > i_{\cancel{b}}(\infty)$ analogously to the special case $q_0 = 0$ above. Specifically, we solve for c^* using the fixed point of Eq. (5) at the maximum infection level (see *Materials and Methods* for the expression). Furthermore, it is in fact the case that $i(\infty)^+$ is stable while $i(\infty)^-$ is unstable. To characterize the stability we note that Eq. (5) can in general be written as the expression of a parabola with a maximum at $[i(\infty)^+ + i(\infty)^-]/2$, as shown in the phase plot in Fig. 6a.

The size of the jump in infection at this transition, Δi_∞, is simply the difference between the stationary fraction of infected individuals for the SIS model with resource constraints,

11

$i(\infty)^+$, and that without resource constraints $i_b(\infty)$ (see Fig. 5b). For the special case $q_0 = 0$ the size of the jump is τ^{-1} and thus the base reproduction number fully determines the jump size (see Fig. 2b and Fig. 5a). However, for $q_0 > 0$ there is generally a dependence on the cost c.

A key point is that Eq. (5) and all of the stability analysis we have carried out is independent of the budget function $f(b)$. Thus, the argument above also shows that the discontinuous emergence of a new endemic infection level holds under the general conditions for a budget function outlined above. In the *Supplementary Information* we show a simple example where the discontinuous behavior is observed for a continuous budget function $f(b)$, further verifying that the discontinuity of the explosive epidemic transition is *not* a consequence of the jump in the Heaviside step function at $b = 0$.

III. DISCUSSION

In summary, we have shown that explosive transitions in the epidemic level in a population occur above a critical healing cost, when the budget for recovery is generated endogenously by the healthy population. Above the critical healing cost the rate of budget generation cannot cover the recovery needs. As a consequence, the infection eventually jumps to a higher level than the standard model would predict. The general behavior of the bSIS model can be summarized in a phase diagram for the mean-field system with two-parameters: healing costs c and effective base reproduction number τ, shown in Figure 6b. Three steady state regimes are found: healthy, low epidemic, and high epidemic. The healthy regime converges to a zero fraction of infected individuals. This regime is observed when $c < c^*$ and $\tau < \tau^*$. The transition to the high epidemic regime always leads to a discontinuous jump in the infection incidence, while the transition between the healthy and epidemic regimes exhibits a continuous change in the infection steady state, as in the classical SIS model. Importantly, an epidemic can break out even when the standard SIS model predicts that the disease will die out without infecting a significant part of the population. This highlights that to accurately assess the risks posed by a disease outbreak, the constraints that healing resources impose need to be taken into account.

We emphasize that the behavior we find is very general, applying across different assumptions about the underlying interaction structure between individuals and the details of

12

the budget function. The scaling exponents describing the transition to the high epidemic regime in a simulated outbreak on a real friendship network are consistent with our analytical calculations that assume a random interaction network. Furthermore, the discontinuous transition to the high epidemic regime exists independent of the specific network topology. This discontinuity indicates the importance of carefully monitoring an outbreak in order to avoid a sudden and uncontrollable transition into the high epidemic regime.

Future work could consider the interplay between the heterogeneous distribution of resource generation and resource demands, across different communities or locations. In this way we could determine how to redistribute the budget, balancing the goal of protecting the greatest number of people in the short-run with the goal of sustaining a high-level of budget contributions. Similarly, we could also determine what locations are strategic points for targeting measures to increase the productivity of healthy individuals.

Finally, variants of the model we consider may also shed light on non-biological contagion processes where budget constraints have a significant impact on the dynamics of the system. A timely example are bankruptcy cascades in the banking sector [39]. Given the high public debt levels in most areas of the world, saving banks and countries suffering from high debt levels might eventually exceed the ability of the tax payer to create the necessary resources.

IV. MATERIALS AND METHODS

A. Friendship network

The friendship network was constructed based on the answers of school students in the United States to an Add-Health questionnaire [40]. From the full set of networks obtained from this data set, we consider the largest component, which consists of 2539 nodes and 20910 edges. Numerical results for the bSIS model on this topology are averages over 3200 simulations. See *Supplementary Information* for a more detailed characterization of the network.

B. Mean-field bSIS model with pure budget mediated recovery

The mean-field bSIS model with pure budget mediated recoveryis as described by Equations (1) and (2) and $q_0 = 0$. As long as $b > 0$, infection levels evolve as in the pure SIS

13

model, and can thus be found by integrating Eq. (1), which gives:

$$i(t) = \frac{i(0)\,(1 - \tau)}{(1 - \tau\,[1 - i(0)])\,e^{(1-\tau)t} - i(0)\tau}\ ,\tag{6}$$

where $i(0)$ is the initial fraction of infected individuals and we have assumed $f(b) = \Theta(b)$.

Similarly, as long as $b \geq 0$, the evolution of the budget is found by integrating Eq. (2):

$$b(t) = \frac{q\,[1 + c\,(q - kp)]\,t + (1 + cq)\log\,[i(t)/i(0)]}{kp}\tag{7}$$

where $i(t)$ is given by Eq. (6). (Here we have set the initial condition $b(0) = 0$, without loss of generality.) The three possible behaviors of the budget shown in Fig. 2b can then be obtained depending on the values of c, q, and kp.

Using Eq. (7) we can determine t^* by solving for $b(t^*) = 0$ (given $c > c^*$). When $\tau \leq 1$ by definition $i(t^*) = i(0)$ and therefore Eq. (7) implies $t^* = 0$. For the case $\tau > 1$, if we assume $t^* \gg 1$, transients decay and the equilibrium epidemic infection level $i(\infty) = 1 - \tau^{-1}$ is reached without budget constraints. In this case

$$t^* = \log\left(\frac{i(\infty)}{i(0)}\right)\tau c^{*2}\left[(c - c^*)^{-1} + \frac{1}{\tau c^*}\right].\tag{8}$$

C.   General Mean-field bSIS model

The bSIS model with $q_0 > 0$ only exhibits interesting behavior when $\tau_{q_0} = kp/q_0 > 1$ (otherwise infection will always die out). Under this assumption, we can set $di(t)/dt = 0$ in Eq. (5) to find the fixed points of the infection dynamics:

$$i(\infty)^{\pm} = \frac{1 + c(kp - q_0) \pm \sqrt{[c(q_0 - kp) - 1]^2 - 4ckp}}{2ckp}.\tag{9}$$

The stability of these fixed points is discussed in the *Methods*. We can see from this expression that this equilibrium infection level is bounded above by the equilibrium of the pure SIS model with recovery rate q_0 (namely $1 - \tau_{q_0}^{-1}$), and approaches it in the limit $c \to \infty$.

We can find the critical cost c^* that guarantees $i(\infty)^+ > i_b(\infty)$ by solving for c^* at the fixed point of Eq. (5) and noting that, as in the case considered in the main text for $q_0 = 0$, the smallest possible c^* for a system is given by the stationary point at the largest infection level reached, $i_{max} = \max_t[i_b(t)]$. This gives:

$$c^* = \frac{1 - i_{max}}{i_{max}[kp(1 - i_{max}) - q]}.\tag{10}$$

In the *Supplementary Information* we present a more detailed characterization of the critical cost c^* that guarantees convergence to the higher infection fixed point.

14

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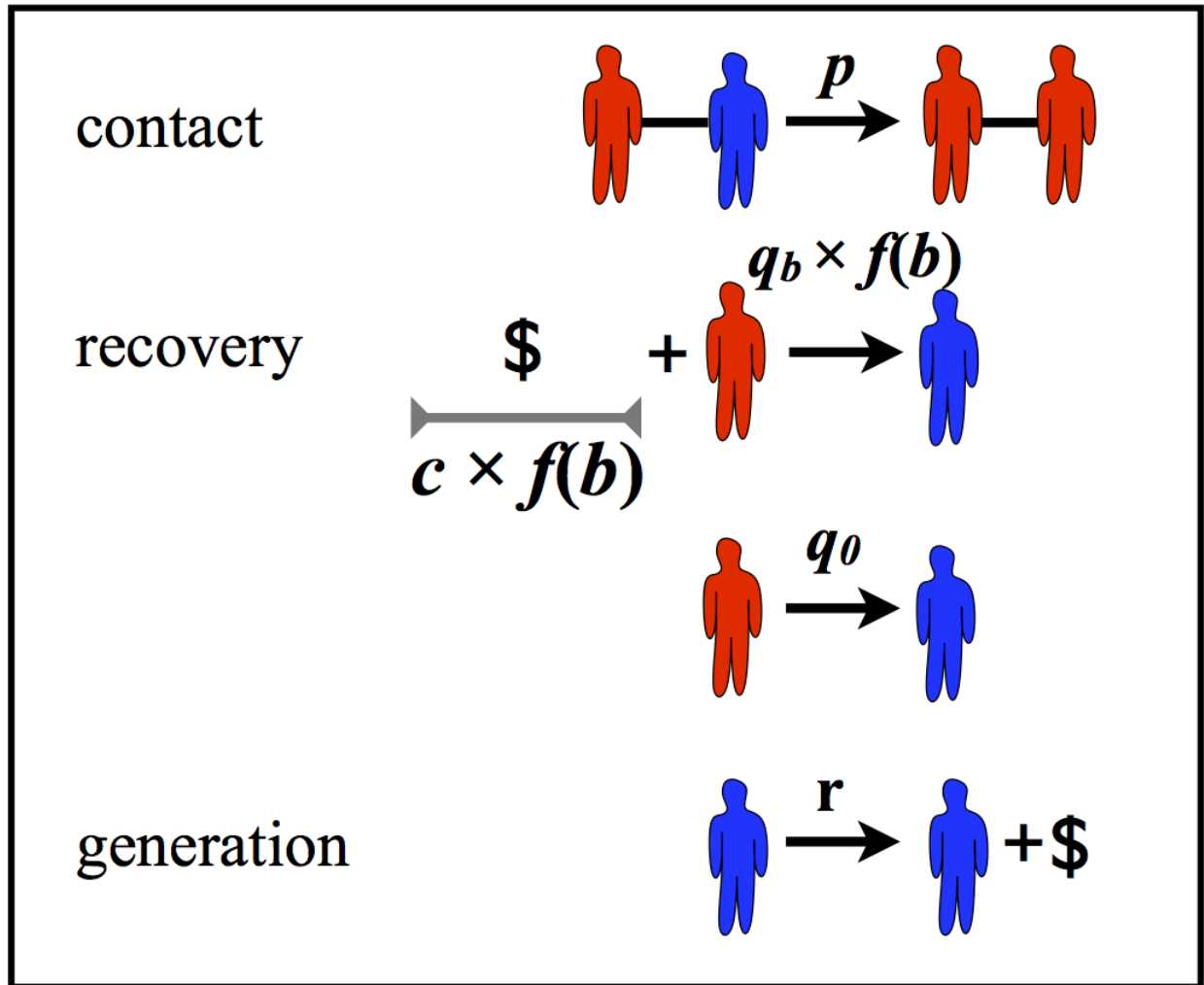
FIG. 1. **The bSIS model.** The four transitions that describe the bSIS model, each occurring at the rate denoted above the transition arrow. As in the SIS model, infected (red) individuals contact and infect susceptible (blue) neighbors at rate p. Infected individuals will recover at a resource mediated recovery rate $q_b f(b)$ if the necessary resources $cf(b)$ are applied, and with an additional baseline rate q_0, independent of resource availability. A unit of budget is generated by each susceptible at a rate r. Without loss of generality we have normalized the model so that one unit of budget is generated by every individual per unit of time and thus $r = 1$.
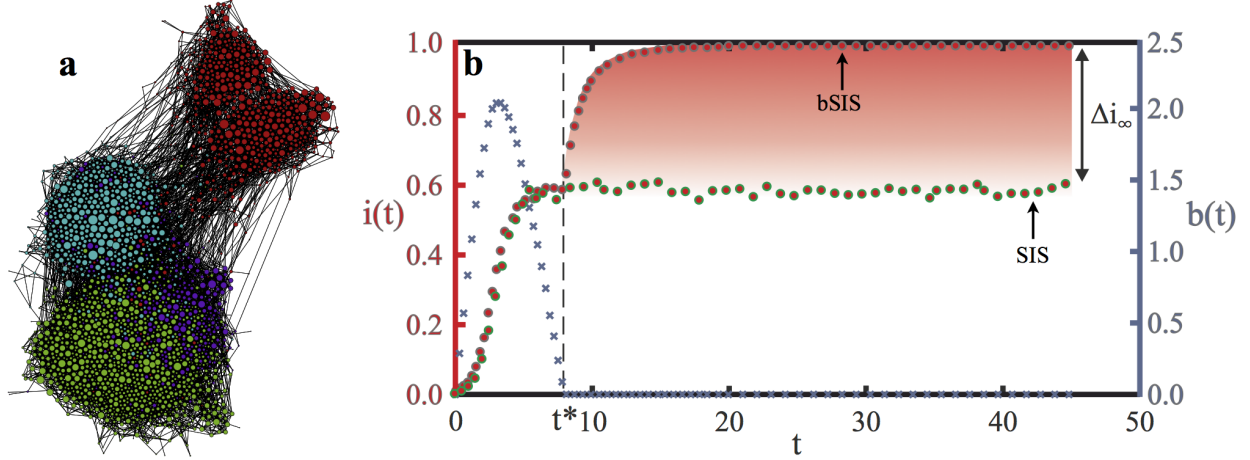
FIG. 2. **Epidemics self-accelerate when resources are endogenously constrained** (**a**) The school friendship network, which exhibits long-range connections, clustering and community structure (colors denote communities found through modularity maximization). (**b**) Evolution of the fraction of infected individuals $i(t)$ (red circles) and the budget $b(t)$ (blue crosses), for healing costs above the critical cost ($c = 2 > c^* \approx 0.833$), on the friendship network. Recovery requires a budget: $q_0 = 0$ and $q_b = 0.8$, and $p = 0.285$. In the pure SIS model the fraction of infected individuals reaches a stationary regime. The bSIS model also initially converges to the same stationary regime until the budget is exhausted at a critical time $t = t^* \approx 7.3$. For $t > t^*$ recovery is not possible anymore, and the infection spreads to all of the population (i.e. $i(\infty) = 1$). The difference in the steady state infection in both models, which is also the size of the jump in the bSIS model due to the budget constraints, is denoted Δi_∞.
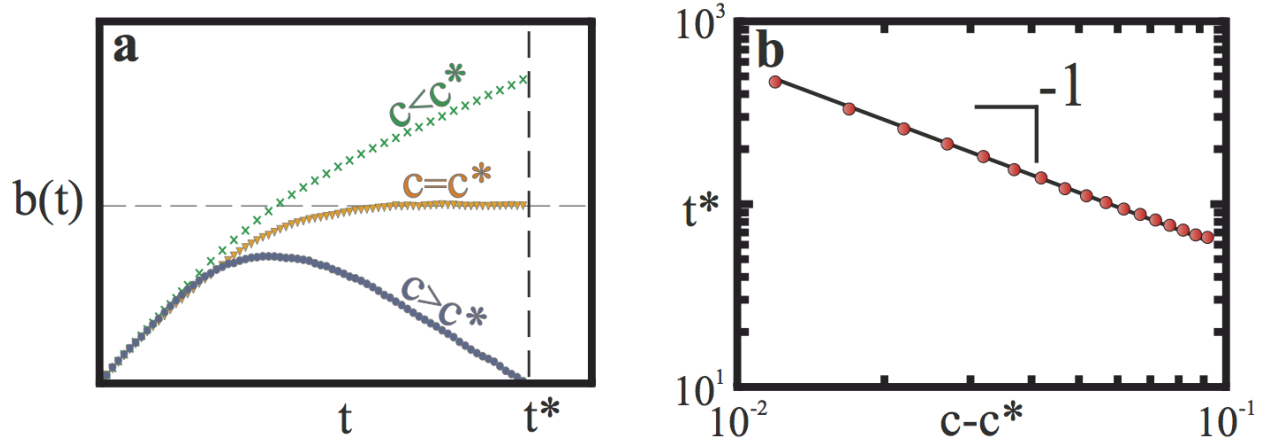
FIG. 3. **The time dynamics of the budget according to the cost of recovery.** (a) Three different regimes of time-dependent budget development $b(t)$, depending on the healing cost c. For healing costs c below the critical cost, c^* (green crosses $c = 0$), the budget is a monotonically increasing function of time. Above the critical cost ($c = 2 > c^*$, blue circles), the budget reaches a maximum and then decreases until it is exhausted at a critical time $t = t^*$, allowing the infection to spread explosively. At the critical cost ($c = c^* = 0.833$, orange inverted triangles), the budget saturates at a non-zero value. (**b**) The critical time scales with the distance to the critical cost according to $t^* \sim (c - c^*)^{-1}$ (averaged over 3600 samples).
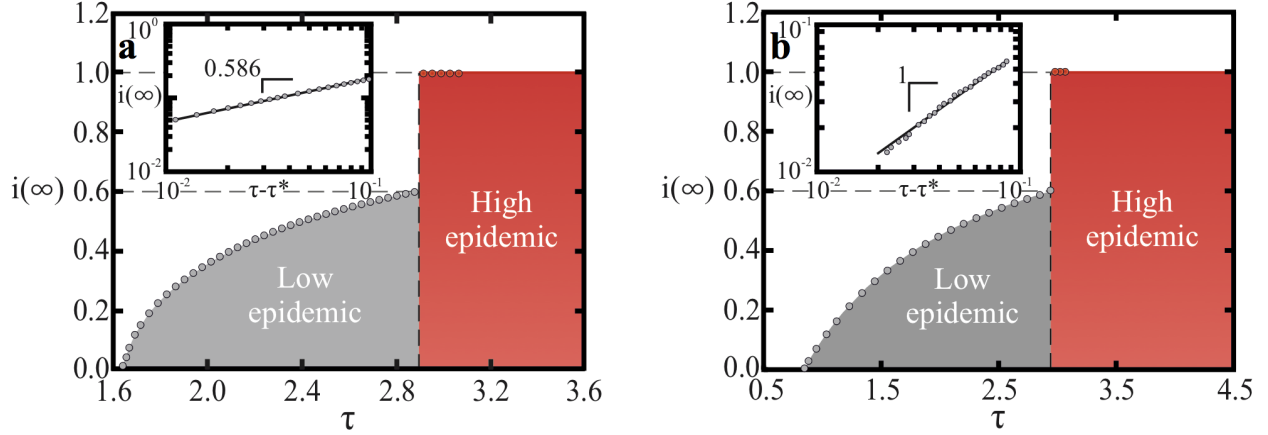
FIG. 4. **Discontinuous transition from the low to the high epidemic regime.** (**a**) Asymptotic fraction of infected individuals $i(\infty)$ on the friendship network, for fixed costs $c = 0.833$ and recovery rate $q = 0.8$ as a function of τ (we vary p and hold k and q fixed). At $p = 0.285$, a discontinuous transition from the low to the high epidemic regime is observed. The inset shows that in the epidemic regime $i(\infty) \sim (\tau - \tau^*)$, just as in the mean-field limit of the SIS model [32]. (**b**) Same as in (a) but on a square lattice with 1024^2 sites. The inset shows the approach of $i(\infty)$ to $\tau^* = 1.6488 \pm 0.0001$, confirming the predicted power-law scaling. The black solid line is a guide to the eye, corresponding to $(c - c^*)^\beta$, where $\beta = 0.586 \pm 0.014$ is the critical exponent of the order parameter for the SIS model on the square lattice. The values of τ^* and β where obtained from Ref. [38]. (Results are averages over 3600 samples.)
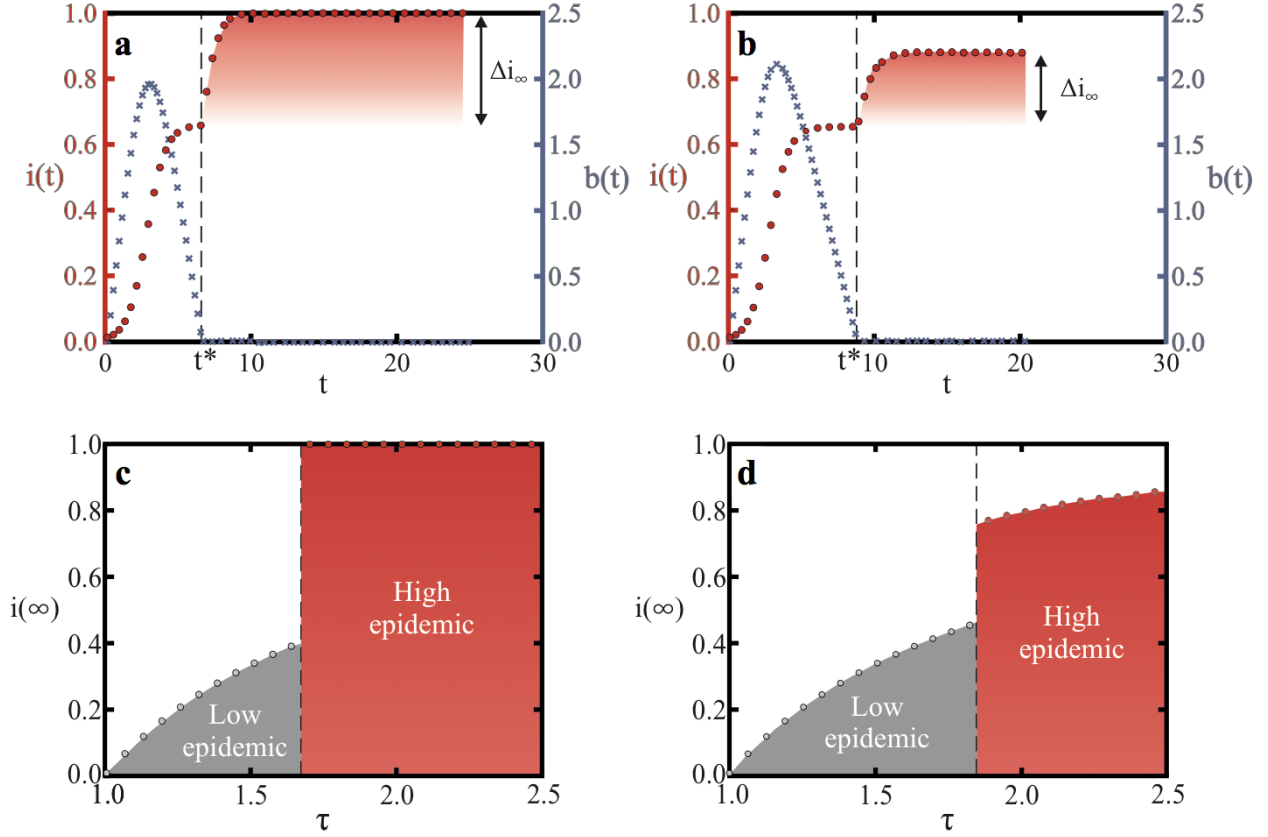
FIG. 5. **Generalizing results to the case with a baseline recovery rate independent of budget** (**a**) Evolution of the fraction of infected individuals $i(t)$ (red circles) and the budget $b(t)$ (blue crosses) in the mean-field model with the same average degree as the friendship network ($\langle k \rangle \approx 8.2355$). All parameters are as in the simulation on the friendship network shown in Fig. 2b: most importantly healing costs are above the critical cost ($c = 2 > c^* \approx 0.833$) and all healing requires a budget contribution ($q_b = 0.8$ and $q_0 = 0$). Clearly the mean-field behavior is very similar to that in the friendship network. (**b**) Mean-field model with a baseline recovery rate $q_0 = 0.2$ and $q_b = 0.6$ ($q = q_0 + q_b = 0.8$ as above). The behavior is similar to (a) but Δi_∞ is smaller due to the baseline recovery. The steady state infection level $i(\infty)$ is bounded from above by $i_{q_0}(\infty)$ (the steady state infection in the pure SIS model with recovery rate $q = q_0 = 0.2$). (**c**) The discontinuous phase transition observed for parameters in (a) as τ increases (by varying p). (**d**) Similarly, the phase transition observed for the model in panel (b). Although $i(\infty)$ increases continuously with τ after the phase transition, the jump at τ^* is clearly discontinuous.
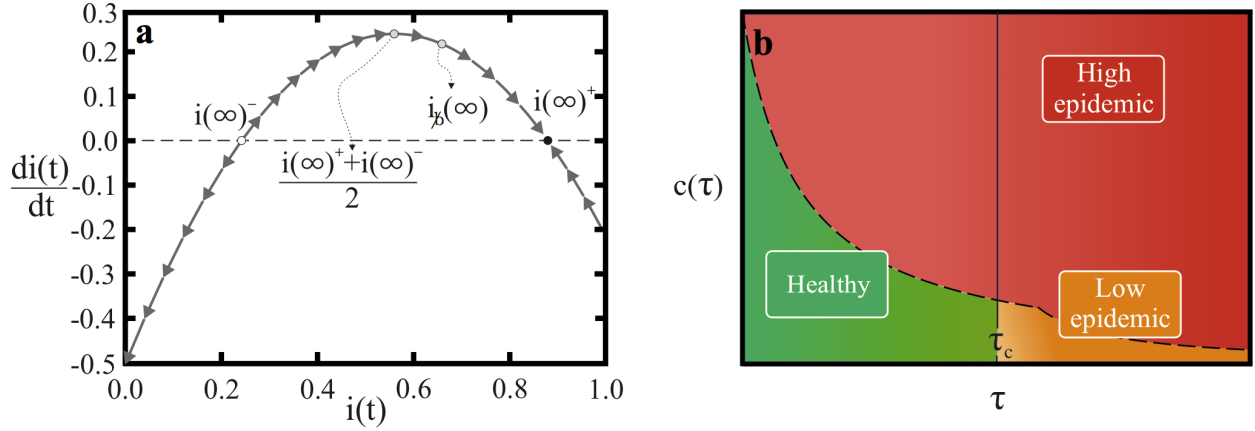
23

FIG. 6. **The explosive epidemic transition to a high epidemic state occurs generically in the bSIS model.** (**a**) The phase space of Eq. (5) and the corresponding stationary states. In this plot the parameters are the same as in Fig. 5b,d. The black dots indicate the stationary states of Eq. (9) and the arrows illustrate the stability of $i(\infty)^+$. In general one can rewrite Eq. (5) to get an equation of a parabola. The equilibrium infection level without the budget restriction is denoted $i_{\not b}(\infty)$, and it is always the case that $i(\infty)^+ > i_{\not b}(\infty)$. (**b**) The general behavior of the bSIS system can be illustrated by a phase diagram of the different regimes depending on healing costs and effective base reproduction number. Three different regimes are found: the *healthy* regime with a zero fraction of infected individuals ($i(\infty) = 0$); the *low epidemic* regime; and the higher infection *high epidemic* regime. As discussed in the text, transitions between the *healthy* and *low epidemic* regimes are always continuous, while transitions to the *high epidemic* regime are discontinuous.