Universal behavior in a generalized model of contagion

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Models of contagion arise broadly both in the biological and social sciences, with applications ranging from the transmission of infectious diseases to the diffusion of innovations and the spread of cultural fads. In this Letter, we introduce a general model of contagion which, by explicitly incorporating memory of past exposures to, for example, an infectious agent, rumor, or new product, includes the main features of existing contagion models and interpolates between them. We obtain exact solutions for a simple version of the model, finding that under general conditions only three classes of collective dynamics exist, two of which correspond to familiar *epidemic threshold* and *critical mass* dynamics, while the third is a distinct intermediate case. We find that for a given length of memory, the class into which a particular system falls is determined by two parameters, each of which ought to be measurable empirically. Our model suggests novel measures for assessing the susceptibility of a population to large contagion events, and also a possible strategy for inhibiting or facilitating them.

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Defined broadly as the transmission of an influence from one individual to another, the concept of contagion occupies an important place both in biology—specifically in mathematical epidemiology [1, 2]—and in the social sciences, where it is in manifested in problems as diverse as the diffusion of innovations [3, 4], the spread of cultural fads [5–7], and the outbreak of political [8] or social [9] unrest.

Despite the wide range of social and biological phenomena to which they have been applied, existing models of contagion typically fall into one of two categories that we delineate in terms of the relationship between successive exposures of a "susceptible" to an "infectious" individual: (1) what we call "Poisson" models, in which successive contacts result in contagion with independent probability p; and (2) "threshold" models, in which the probability of infection changes rapidly from low to high as a critical number of simultaneous exposures is exceeded (thus the effect of any single exposure depends strongly on the number of other exposures). The SIR model [10], the canonical model of biological contagion, is an example of a Poisson model, as is the oft-cited Bass model [3] from the diffusion of innovations literature. By contrast, numerous models in sociology [9], economics [11], and political science [12], are explicitly threshold models; while others still [13–15] embed thresholds implicitly through the relative costs and benefits of one action versus another.

None of these models, however, treat the interdependencies between exposures themselves as an object of study—rather they are simply assumed to either exist or not exist—hence their effects on the collective dynamics of contagion are unknown. Furthermore, if, as we show below, these effects turn out to be considerable, existing models provide no way to determine the conditions under which one kind of collective behavior or another should be expected.

In this Letter, we explore a generalized model of contagion that, by introducing memory of past exposures to a contagious influence, generalizes and interpolates between Poisson and threshold models of contagion. Our model is defined as follows. Consider a population of N individuals, each of which is in one of three states S (susceptible), I (infected), or R (removed). At each time step t, each individual i comes into contact with one other individual j, drawn randomly from the population. If i is susceptible and j is infected then, with probability p, i receives a positive dose $d_i(t)$, drawn randomly from some distribution of dose size $f(d)$; otherwise, $d_i(t) = 0$. Each individual maintains a memory of doses received over the previous T time steps, recording a cumulative dose $D_i(t) = \sum_{t'=t-T+1}^{t} d_i(t')$. Susceptible individuals become infected if $D_i(t) \geq d_i^*$ where d_i^* (i's *dose threshold*) is drawn randomly at $t = 0$ from a distribution $g(d^*)$, and remains fixed thereafter. The probability that a susceptible individual who encounters $K \leq T$ infected individuals in T time steps will themselves become infected

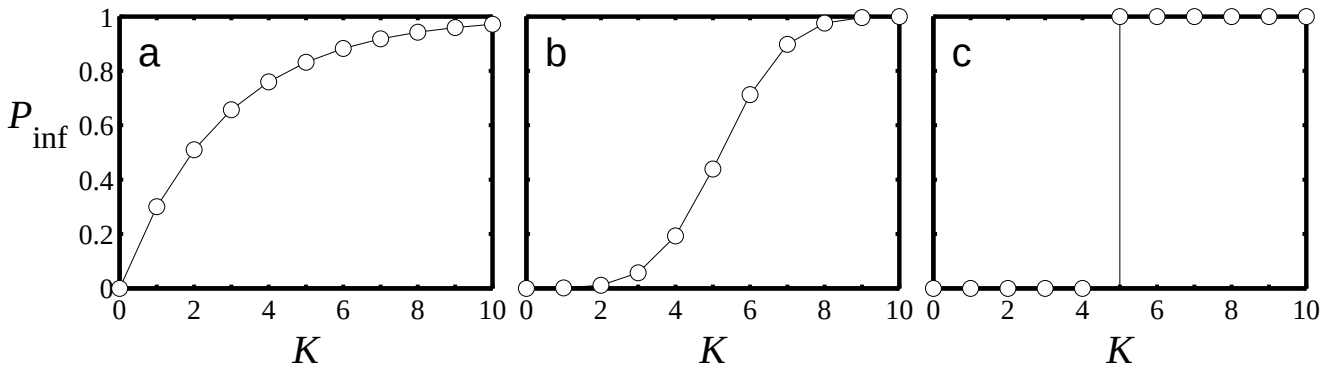

FIG. 1: Dose response curves for $T = 10$ (see Eq. (1)): (a) Poisson (e.g., SIR-type) model: probability of receiving a positive dose by contacting an infective $p = 0.3$, distribution of dose sizes $f(d) = \delta(d-1)$, and distribution of individual dose thresholds $g(d^*) = \delta(d^* - 1)$; (b) Stochastic threshold model: $p = 1$, $f(d)$ is distributed lognormally with unit mean and variance 0.4333, and $g(d^*) = \delta(d^* - 5)$; and (c) Deterministic threshold model: $p = 1$, $f(d) = \delta(d-1)$, and $g(d^*) = \delta(d^* - 5)$.

is therefore

$$P_{\text{inf}}(K) = \sum_{k=1}^{K} \binom{K}{k} p^k (1-p)^{K-k} P_k, \tag{1}$$

where

$$P_k = \int_0^\infty \mathrm{d}d^* g(d^*) P\left(\textstyle\sum_{i=1}^k d_i \geq d^*\right) \tag{2}$$

is the average fraction of individuals infected after receiving k positive doses in T time steps, and $P(\sum_{i=1}^k d_i \geq d^*)$ is the probability that the sum of k doses drawn from $f(d)$ exceeds a given d^*.

Equation (1) can be thought of as an average dose-response relationship [16] for the population in question. Figure 1 displays three examples of Eq. (1) for different choices of p, $f(d)$, and $g(d_*)$. When all doses $d_i = \bar{d}$ are identical, all members of the population have the same threshold $d^* = \bar{d}$ and $p < 1$, then Eq. (1) reduces to the standard SIR model, Fig. 1(a); and when $p = 1$ and $d^* > \bar{d}$, it is equivalent either to a stochastic [13, 15], or deterministic [9, 11, 17] threshold model, depending on whether doses are allowed to vary, Fig. 1(b), or are identical, Fig. 1(c). More complicated choices of $f(d)$ and $g(d^*)$ correspond to many other kinds of models that incorporate varying degrees of interdependency between contagion events and also heterogeneity across individuals.

Once infected, individuals may recover with probability r if $D_i(t)$ falls once more below d^* (otherwise they remain infected), and recovered individuals become re-susceptible with probability ρ. While the resulting dynamics is, in general, quite complex, in the special case of $\rho = 1$ and $r = 1$ (analogous to so-called SIS-type [18, 19] models with instantaneous recovery), we can write down an equation for the steady-state fraction of infectives (fixed points) in the population [27]:

$$\phi^* = \sum_{k=1}^{T} \binom{T}{k} (p\phi^*)^k (1 - p\phi^*)^{T-k} P_k, \tag{3}$$

with P_k as defined by Eq. (2).

Exact solutions of Eq. (3) may be obtained numerically (i.e., to arbitrary precision) and agree with results from our simulations [20]. Moreover, we are able to determine analytically that the equilibrium behavior of our generalized model falls into one of only three universal classes, examples of which are given in Fig. 2. We define these classes by the behavior of the fixed point curves around a single transcritical bifurcation [21], which is present for all models and located at $p = p_c = 1/(TP_1)$ and $\phi^* = 0$. We find that for a given value of the memory parameter T, the class into which a particular model falls depends only on the values of two quantities: P_1 and P_2, the probability that an individual will become infected as a result of one and two exposures respectively. The three classes of behavior and their associated conditions are as follows.

Class I: epidemic threshold models. When $P_1 \geq P_2/2$, we find the equilibria of Equation 2 fall on a bifurcation curve similar to that shown in Fig. 2(a), in which the sole bifurcation is the transcritical one at $p = p_c < 1$ (analogous to a continuous phase transition [22]). When $p \leq p_c$, a stable equilibrium exists at $\phi^* = 0$ (i.e., all initial seeds die out exponentially fast), and when $p > p_c$, $\phi^* > 0$ implying that a finite fraction of the population will become infected (i.e., an epidemic will occur). Thus the dynamics of models in class I is qualitatively equivalent to that of SIR-type models in which p_c, sometimes called the epidemic threshold [23], determines the critical value of the infectiousness p required in order for an initial seed of infectives to trigger an epidemic. We therefore call models in class I *epidemic threshold* models.

Class II: vanishing critical mass models. When $P_2/2 > P_1 \geq 1/T$, we find, as shown in Fig. 2(b), a change in

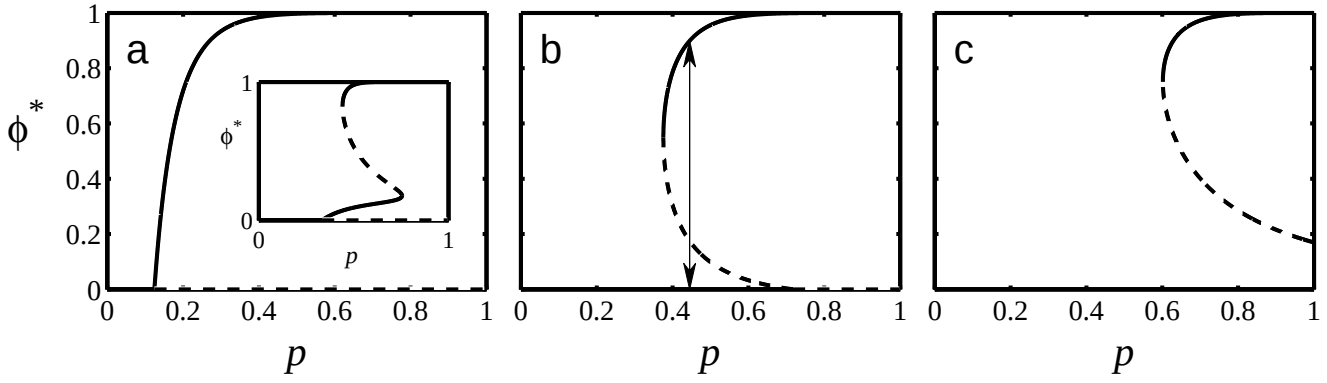

FIG. 2: Example fixed point curves for the three basic classes of contagion models (all curves are obtained numerically using the closed form expression of Eq. (3) and agree with results from simulations (not shown) [20]): a) Class I: Epidemic threshold, b) Class II: Vanishing critical mass, and c) Class III: Pure critical mass. Dose sizes are lognormally distributed with mean 1 and variance 0.433, $T = 10$, and thresholds are uniformly set at a) $d^* = 0.5$, b) $d^* = 1.6$, and c) $d^* = 3$. In the class II example of (b), trajectories of two initial conditions are indicated by arrows. For $p = 0.445$ and $\phi(0) = 0.174$, the contagion fails to persist while for the same p and a slightly higher initial level of infection, $\phi(0) = 0.175$, the contagion is sustained with 90% of individuals eventually infected. At the transition between classes I and II, the saddle-node and transcritical bifurcations coincide yielding a continuous phase transition distinct in nature from those of class I. The behavior of the fixed point curve near $p = p_c$ is $\phi^* \propto (p - p_c)^{1/2}$ rather than $\phi^* \propto (p - p_c)^1$, though in highly special cases the exponent is lower [20]. Inset in (a): An example of a more complicated fixed point diagram. Here, $T = 20$, dose size is set to unity, $f(d) = \delta(d-1)$, and $d^* = 1$ with probability 0.15 and 6 with probability 0.85. In principle, systems with more bifurcations are possible although unlikely since a strongly multimodal distribution of thresholds and/or dose sizes is required.

the nature of the transcritical bifurcation. Whereas for class I models, a stable fixed point curve emanates from $(p_c, 0)$ with positive slope, class II models have an unstable fixed point curve entering the transcritical bifurcation with a negative slope. Accompanying this is the appearance of a saddle-node bifurcation [21] at $p = p_b < p_c$ and $\phi = \phi_b^* > 0$ (the system now exhibits a first-order phase transition). The solid (dashed) lines in Fig. 2(b) correspond to the position of stable (unstable) equilibria respectively; thus there exists a region in p in which two stable equilibria coexist, separated by an unstable equilibrium. In other words, as shown by the arrows in Fig. 2(b), if the initial seed $\phi(0)$ falls below the unstable equilibrium, then the contagion will die out; whereas if it falls above the unstable equilibrium, then a large fraction of the population (corresponding to the upper stable equilibrium) will be infected (i.e., the system is metastable [22]). Thus infections in class II require a "critical mass" [24] to succeed. Because the size of this critical mass decreases to zero for $p < 1$, we call this class of models *vanishing critical mass* models, where we note that the sensitivity to initial conditions implicit in critical mass dynamics is absent entirely from models in class I; hence the two classes are qualitatively distinct in terms of their equilibrium behavior.

Class III: pure critical mass models. When $1/T > P_1$, the position of the transcritical bifurcation $p_c > 1$; hence the only bifurcation potentially remaining in the interval $0 \leq p \leq 1$ is the saddle-node bifurcation, see Fig. 2(c). Thus in the manner of first-order phase transitions, a finite critical mass is always required (i.e., for all p) in order for an initial seed not to die out. As a result, we call models in this class *pure critical mass* models, where our classification of pure and vanishing critical mass models includes the dynamics of familiar threshold models [9, 12, 17], but is more general.

For some choices of $f(d)$ and $g(d^*)$, additional equilibria are possible, and correspond to additional saddle-node bifurcations in Figure 2. Investigations of Eq. (3) and numerical simulations [20] indicate that for additional equilibria to appear, one or both of $f(d)$ and $g(d^*)$ must be multimodal, with widely separated peaks. For example, the inset of Fig. 2(a) shows the solutions of Eq. (3), where 15% of the population have $d^* = 1$ and 85% have $d^* = 6$. In addition to the standard Class I transcritical bifurcation, the inset shows a new saddle-node bifurcation. Extra saddle-node bifurcations generated in this manner, do not affect either the position or nature of the transcritical bifurcation, which continues to depend only on P_1 and P_2. Hence our basic classification scheme, outlined above, is preserved for arbitrary $f(d)$ and $g(d^*)$.

The existence of three basic classes, and also simple conditions that determine which class a particular contagion model belongs to, has interesting implications for understanding and possibly influencing the collective dynamics of contagion, whether biological or social. First, the result dramatically reduces the effective complexity of the generalized model by showing that the qual-

itative dynamics are independent of many of the details of the particular population [i.e., $f(d)$ and $g(d^*)$]; hence our use of the term "universal classes". Equally important, however, is that not all models fall into a single class; that is, not all kinds of contagion are the same. Furthermore, the three classes are non-degenerate in the rough sense that the conditions for each occupy broad regions of the parameter space. This result is not to suggest that some kinds of contagion are not more likely than others, but it does suggest that without empirical evidence about the relevant P_1 and P_2, little can be concluded about the corresponding collective dynamics.

Because neither the mathematical epidemiology literature nor the micro-organismal dose-response literatures have tended to question the assumption that infection is a Poisson process, experimental evidence for or against the assumption is limited. In the context of biological contagion, therefore, our model suggests a very clear test to validate or refute the notion of interdependencies between exposures, and also specifies the strength of interdependencies required in order for them to be considered important with respect to the collective dynamics. Specifically, if $P_1 > P_2/2$ then the assumption of independence between successive exposures can be considered valid, whether or not it is precisely true, in the sense that the same qualitative behavior arises regardless. If, however, $P_1 < P_2/2$ for some infectious agents or in some populations, then the interdependencies cannot be ignored, as their effect, in terms of the equilibrium states of the collective dynamics, may be dramatic.

In the context of social contagion, our model has a different implication: that under very general, and one might argue likely, conditions, SIR-type models such as the Bass model that do not include memory or interdependencies between subsequent exposures, are incapable of capturing even the basic features of contagion dynamics. However, there are also likely to be some applications in which the independence assumption does turn out to be valid—that is, threshold models are also unlikely to be universally appropriate models of social contagion—where the key point is that our model provides precise conditions under which one state or the other will pertain.

A related point is that the conditions on P_1 and P_2 that separate the three classes of behavior suggest novel intervention strategies for suppressing, or alternatively stimulating, global contagion. Assuming that an individual's threshold d_i^* can be manipulated—for example, increased (in a biological sense) by better preventative health treatment, or decreased (in a social sense) by exerting some financial, social, or cultural influence—then our results suggest that relatively minor manipulations (e.g., altering P_1 and P_2 to shift p_c while leaving the class of contagion unchanged) can have a dramatic impact on the ability of a small initial seed to trigger a global contagion event (i.e., the accessibility of a non-zero stable equilibrium) as well as on the size of such an event if it occurs. We note that manipulating P_1 and P_2 is not equivalent to manipulating p since p determines the relevant point on the bifurcation curve, whereas P_1 and P_2 determine the shape of the curve itself; one cannot subsume temporal interdependencies within the independence assumption simply by lowering the per-event probability of transmission. Finally, we note that while recent theoretical work on controlling disease epidemics, or by contrast stimulating social contagion, has focused on individuals of exceptional influence (e.g., so-called "super-spreaders" [25, 26], and "opinion leaders" [4]), our model suggests that it could be the most easily *influenced* individuals (i.e., those contributing to P_1) who have the greatest impact on the dynamics of contagion.

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