Slightly generalized Generalized Contagion: Unifying simple models of biological and social spreading

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We motivate and explore the basic features of generalized contagion, a model mechanism that unifies fundamental models of biological and social contagion. Generalized contagion builds on the elementary observation that spreading and contagion of all kinds involve some form of system memory. We discuss the three main classes of systems that generalized contagion affords, resembling: simple biological contagion; critical mass contagion of social phenomena; and an intermediate, and explosive, vanishing critical mass contagion. We also present a simple explanation of the global spreading condition in the context of a small seed of infected individuals.

I. INTRODUCTION

Spreading, construed fully, is everywhere: the entropically aspirant diffusive relaxation of all systems; wave motion, for which ubiquitous is assigned with no overstatement; in the propagation of earthquakes; the expansion of species range, so often involving people; power blackouts, now able to affect large fractions of the world population through system growth; the repeated bane of global pandemics; economic prosperity and misery; and the talk of the famously talked about. And understanding how myriad entities spread between people—from diseases to stories, both true and false—is central to our scientific understanding of large populations.

Used for good, as the trope goes, a deep knowledge of contagion mechanisms—contagion science—is necessary to help in our collective efforts to produce a world where individuals can flourish. Used for bad, a path scientific knowledge always offers, malefactors will be empowered in the persuasion and manipulation of populations or the breaking of financial systems. To prevent negative and catastrophic outcomes, contagion science should be able to provide us with algorithms for system defense.

There remain many open questions on contagion. How many types of spreading and contagion mechanisms are there? How can we identify and categorize real-world contagions? But we have only recently moved from the data-scarce period of studying social phenomena to the start of the data-rich stage, and contagion science is still very much developing.

Our goal in this piece is constrained to revisiting our 2004 revisiting of basic mathematical models of contagion surrounding one question [1, 2]: Can we connect models of disease-like and social contagion?

We call the process we constructed for this objective ‘generalized contagion’. We will give a straightforward explanation of the model here and discuss its most important features.

An incidental contribution with generalized contagion was to make memory a primary ingredient. For contagion, memory comes in many forms, for example, in the development of protection against an infectious disease through an immune response, or through recalling past exposures to some kind of social influence. The core models of biological and social contagion incorporate only the simplest kind of memory, that of the present state.

In proceeding, we first outline the independent and interdependent interaction models of biological and social contagion. Apart from standing as the footing of our generalized model, we will also preserve certain framings and notations. We then describe our model of generalized contagion and discuss the three universality classes of systems identified in the context of small seeds leading to global spreading.

II. INDEPENDENT INTERACTION MODELS OF BIOLOGICAL CONTAGION

In mathematical epidemiology, the standard model [3] was first put forward in the 1920s by Reed and Frost and formalized by Kermack and McKendrick [4–6]. These models came to be generally referred to as SIR models in reference to the three epidemiological states:

• Susceptible;
• Infective (or Infectious);
• Recovered (or Removed or Refractory).

Individuals cycle through the states \( S \) to \( I \) to \( R \) (and then back to \( S \) for an SIRS model). The behavior of these initial models was described by differential equations but...
can be easily realized as a discrete time system, and we will use the latter framework for our generalized model. SIR models are also mass action type models, meaning individuals are represented as normalized fractions of a population which randomly interact with each other.

To connect notation across different models, we will write the fractions in the three states as $S_t$, $I_t$ (normally $I_t$), and $R_t$. We must have the constraint $S_t + I_t + R_t = 1$. There is no memory in these systems other than the current balance of Susceptibles, Infectives, and Recovereds.

Fig. 1 shows an example automata for the independent interaction model when time is discrete. From the point of view of an individual agent in a discrete time SIR system, they interact independently, at each time step connecting with a Susceptible, Infective, or Recovered. The probabilities of each interaction are equal to the normalized fractions $S_t$, $\phi_t$, and $R_t$. When Susceptibles interact with Infectives (occurring with probability $\phi_t$), they themselves become Infective with probability $p$. Regardless of their interactions, Infectives recover with a probability $r$ and Recovereds become Susceptibles with probability $\rho$ (for SIR models, $\rho = 0$, while for SIRS models, $\rho > 0$).

A traditionally key quantity in mathematical epidemiology is the Reproduction Number $R_0$ [which is terrible notation given we already have state $R$ and $R_0$]. The Reproduction Number is the expected number of infected individuals resulting from the introduction of a single initial infective. The Reproduction Number is easily interpreted and leads to an Epidemic threshold: If $R_0 > 1$, an 'epidemic' occurs. As with many complex systems, the focus on a single number as a diagnostic is always fraught, and the Reproduction Number ultimately combines too many aspects of the disease itself and population interaction patterns, rendering it a deceptive measure [7]. Nevertheless, for simple models $R_0$ is important and the notion of an Epidemic Threshold is more generally essential.

For our simple discrete model, we can compute $R_0$ easily. We introduce one Infective into a randomly mixing population of Susceptibles. At time $t = 0$, this single Infective randomly bumps into a Susceptible who is infected with probability $p$. The single Infective remains infected with probability $1 - r$ at time $t$, having attempted to infect $t$ Susceptibles by this point. The expected number infected by original Infective is therefore:

$$R_0 = p + (1 - r)p + (1 - r)^2p + (1 - r)^3p + \ldots = p \frac{1}{1 - (1 - r)} = p/r,$$

and the disease spreads in this system if

$$R_0 = p/r > 1.$$  \hspace{1cm} (2)

Fig. 2 shows an example of epidemic threshold from our elementary SIR model where the tunable parameter is the Reproduction Number $R_0 = p/r$. The final fraction infected exhibits a continuous phase transition (technically a transcritical bifurcation [8]). The epidemic threshold is a powerful story arising from a simple model.
III. INTERDEPENDENT INTERACTION MODELS OF SOCIAL CONTAGION

In spite of the basic SIR model’s failings to represent biological contagion accurately in all cases and particularly at large scales, it has enjoyed a long tenure. There have also been overly courageous attempts to use SIR and its sibling models beyond disease spreading including the adoption of ideas and beliefs, the spread of rumors, the diffusion of innovations, and the spread of fanatical behavior.

And while some kinds of social contagion may be disease-like, it is clearly of a different nature for the most part. One of the major departures is due to the fact that people take in information from potentially many sources and weigh their inputs relatively. This observation gives rise to the notion of thresholds, first used in modeling in the early 1970s by Schelling in his efforts to understand segregation (the so-called tipping of neighborhoods, and the origin of “Tipping Point”). Schelling’s model played out (literally) on a chessboard and was manifestly spatial.

Later in the same decade and inspired in part by Schelling’s work, Granovetter produced a distilled mass action threshold model which would become famous in its own right. While social contagion is arguably more multifaceted that biological contagion, Granovetter’s model will serve as our elemental model here.

An individual in Granovetter’s model may be framed as having a choice of adopting a behavior or not based on their perception of that behavior’s popularity. Each individual $i$ has a threshold $d_i^* \in [0, 1]$ drawn from a population-level threshold distribution $P^{(thr)}$ at $t = 0$. We can preserve the SIR model framing of two states: $S$ and $I$, with infectives being those who have adopted the behavior. We will continue to use $\phi_t$ as the fraction individuals who are infected.

At each time step, if individual $i$ observes the fraction $I$ of the total population expressing the behavior as meeting or exceeding their threshold $d_i^*$, then they adopt the behavior. The system iterates forward, potentially reaching an asymptotic state.

Without any spatial structure, all of the interesting dynamics of Granovetter’s model is generated purely by the threshold distribution $P^{(thr)}$. We are in fact in the realm of maps of the interval, the territory where so many extraordinary findings have been made for dynamical systems and chaos. The time evolution of Granovetter’s model can be written down as:

$$\phi_{t+1} = \int_0^{\phi_t} P(u)^{\text{thr}} \, du. \quad (3)$$

The fraction infected in the next time step $\phi_{t+1}$ will be exactly the fraction whose threshold is exceeded by the current fraction infected $\phi_t$.

Writing $P(u)^{\text{thr}}$ as the cumulative function of $P(u)^{\text{thr}}$, we have, compactly, that

$$\phi_{t+1} = P(u)^{\text{thr}} \geq \phi_t. \quad (4)$$

The dynamics of Granovetter’s model are thus inscribed in $P(u)^{\text{thr}}$ particularly in $P(u)^{\text{thr}}$’s fixed points and relative slopes. As an example, Fig. 3 shows how Granovetter’s model may represent a critical mass phenomenon. Fig. 3A gives the distribution $P(u)^{\text{thr}}$ of individual thresholds showing a middle tendency. There are very few extremely gullible people ($d^* \simeq 0$) and very difficult to influence ones ($d^* \simeq 1$). In Fig. 3B, the cumulative function with some example cobweb iterates show that if the initial fraction infected is above the internal fixed point, the fraction adopting the behavior rapidly approaches 1, while any initial fraction starting below the fixed point will see the behavior die out. The initial adoption level $\phi_0$ must be generated by an exogenous mechanism (e.g., education, marketing) and then the purely imitative dynamics of the system take off.

Granovetter’s model and its variants are rich in dynamics and avenues of analysis. In reintroducing spatial interactions, Watts transported Granovetter’s model to random networks showing that limiting an individual’s awareness to a small set of neighbors on a network could lead to large-scale, potentially catastrophic and unexpected spreading. And in moving to more structured, socially realistic networks, even more surprising dynamics open up as possibilities.

IV. GENERALIZED CONTAGION MODEL

The SIR and threshold models are of course intended to be simple, extracting the most amount of story from the least amount of stage setting. But let’s list some standard “I have two comments”-type complaints anyway. As we have trumpeted, both models involve no memory other than of the current state traditional disease models assume independence of infectious events. Threshold models only involve proportions: $17/73 \equiv 170/730$. 
Threshold models also ignore the exact sequence of influences and assume immediate and repeated polling. Other issues applying to both models, and ones that we will not attend to here, include the choice between continuous and discrete time, synchronous updating for discrete time models, and the dominant assertion of random mixing populations (even so, network effects are only part of the story as media provides population-scale and sub-population scale signals). (Standard random scientist issue: “You did not cite my work [which you will find out is not related].”)

We would like to bring these basic models of biological and social contagion together, and, if this is possible, see if we can gain some new knowledge about contagion processes in general. Adding memory will be the way forward. Memory has been successfully incorporated into other kinds of social contagion models with a view to modeling real world behavior online [24, 25].

We explain generalized contagion in the context of a random mixing model acting on a population of N individuals. We will again have the three states S, I, and R, for susceptibles, infectives, and recovereds.

The major variation on the previous models is that each individual has a fixed memory length T drawn from a distribution $P^{(mem)}$ with $1 \leq T \leq T_{\text{max}}$. In [1] and [2], T was the same for all individuals. At all times, individual i possesses a record of their last $T_i$ interactions, a kind of ticker tape memory. Each entry in individual i’s memory will be either zero or a dose received from a successful interaction with an infective (details below).

As for Granovetter’s model, we allow for a general threshold distribution, $P^{(thr)}$. All nodes randomly select a threshold $d^*$ using $P^{(thr)}$, and thresholds remain fixed. Both memory and thresholds could be made to vary with time though we do not do this here.

Here’s the game play for each step.

At each time step, regardless of their current state, each individual i will interact with a randomly chosen individual $i'$ from the population. Next:

1. Individual $i'$ will be an infective with probability $\phi_i$, the current fraction of infectives.
   
   (a) With probability $p$, a dose is successfully transmitted to $i$—an exposure. The dose size $d$ will be drawn from a distribution $P^{(dose)}$.
   
   (b) With probability $1 - p$, $i$ will not be exposed and they will record a dose size $d = 0$.

2. Individual $i'$ will not be an infective with probability $1 - \phi_i$ and $i$ will record $d = 0$ in its memory.

For the SIR model, $p$ was the probability of a successful infection whereas now it is the probability of a successful transmission of a dose which is in turn probabilistic.

Node i’s updates its current dosage level $D_{t,i}$ as the sum of its last $T_i$ doses:

$$D_{t,i} = \sum_{i'=t-T_i+1}^{t} d_{t,i}.$$  \hspace{1cm} (5)

We can now define transition probabilities for individuals in each of the three states. As shown in Fig. 4:

- **S $\Rightarrow$ I**: Infection occurs if individual $i$’s ‘threshold’ is exceeded:

  $$D_{t,i} \geq d^*_i.$$  \hspace{1cm} (6)

- **I $\Rightarrow$ R**: Only if $D_{t,i} < d^*_i$, individual $i$ may recover to state R with probability $r$.

- **R $\Rightarrow$ S**: An individual $i$ may become susceptible again with probability $\rho$. A detail here is that we allow nodes that arrive in state R an immediate chance of returning to S in the same time step. Nodes in state R are immune and will remain in state R even if their dosage level $D_{t,i}$ exceeds their threshold.

V. ANALYSIS

We now perform some basic analyses of the generalized contagion model with a focus on determining the potential for a small seed to lead to a global spreading event, and characterizing the abruptness of that spreading if it is possible. In doing so, we will show how the dynamics of the SIR and threshold models are contained within that of generalized contagion.

Expanding on the results of [1, 2], the key quantity for our analysis is the probability that a randomly selected threshold $d^*$ will be exceeded by $k$ randomly selected doses drawn from $P^{(dose)}$. Using the notation $P_k^{(\text{inf})}$ we have

$$P_k^{(\text{inf})} = \int_0^\infty \, dd^* \, P_k^{(\text{thr})} \Pr \left( \sum_{j=1}^{k} d_j \geq d^* \right).$$  \hspace{1cm} (7)
The integral is over all thresholds $d^*$, and the probability in the integrand is the cumulative distribution of the convolution of $k$ copies of the dose distribution $P^{(dose)}$.

The probabilities $P_1^{(inf)}$ and $P_2^{(inf)}$ will prove to be essential. In particular, $P_1^{(inf)}$, the probability that one randomly chosen dose will exceed one randomly chosen threshold will determine if SIR-like dynamics are possible. The quantity $P_1^{(inf)}$ can be interpreted as the population fraction of the most “vulnerable” individuals [19]. Whatever the length of memory $T$ of these individuals, they typically require only one dose to become infected, and their high susceptibility enables the contagion to spread. This is a harder story to see and many are readily taken by the simpler, naive ones of “super-spreaders” and “influentials.”

We will consider the SIS version, $\rho = 1$, and the case of immediate recovery once an individual dosage drops below its threshold, $r = 1$. Although more difficult, some analytic work can be carried out if these probabilities are reduced below 1 (many variations are explored in [2]), and, of course, simulations can always be readily performed.

As with many dynamical systems problems, we are able to determine the main features of the $\rho = r = 1$ generalized contagion system by examining the system’s fixed points which follow from the system’s update equation:

$$\phi_{t+1} = \frac{\gamma}{\sum_{T=1}^{T_{\text{max}}} P_T^{(\text{mem})}} \sum_{k=1}^{T} \left( \frac{T}{k} \right) (p \phi_{t})^k (1 - p \phi_{t})^{T-k} P_k^{(\text{inf})}. \quad (8)$$

Reading through the right hand side of this fixed point equation, we first have the probability that a randomly chosen individual has a memory of length $T$, $P_T^{(\text{mem})}$. The inner sum then computes the probability that an individual with memory of length $T$’s threshold is exceeded after receiving all possible numbers of positive doses, $k = 1$ to $k = T$.

To find a closed form expression for the fixed points of the system, we set $\phi_{t+1} = \phi_*$:

$$\phi_* = \frac{\gamma}{\sum_{T=1}^{T_{\text{max}}} P_T^{(\text{mem})}} \sum_{k=1}^{T} \left( \frac{T}{k} \right) (p \phi_*)^k (1 - p \phi_*)^{T-k} P_k^{(\text{inf})}. \quad (9)$$

In general, curves for $\phi_*$ as a function of the exposure probability $p$ will need to be determined numerically. However, for the question of whether a small seed may lead to a global spreading event or not, we can use Eq. (9) to find universal results.

Expanding Eq. (9) for $\phi_*$ near 0 we obtain:

$$\phi_* = \frac{\gamma}{\sum_{T=1}^{T_{\text{max}}} P_T^{(\text{mem})}} T P_1^{(\text{inf})} + O(\phi_*^2). \quad (10)$$

Taking $\phi_* \to 0$, we find the critical exposure probability for the system is therefore given by

$$p_c = \frac{1}{\langle T \rangle P_1^{(\text{inf})}}, \quad (11)$$

where $(T) = \sum_{T=1}^{T_{\text{max}}} T P_T^{(\text{mem})}$ is the average memory length (if all individuals have a memory of uniform length $T_*$, as assumed in [1] and [2]. Eq. (11) reduces to $p_c = 1/[T_* P_1^{(\text{inf})}]$.) We interpret $p_c$ in the same way as the epidemic threshold of the SIR model. Global spreading from small seeds will occur if $p > p_c$, and this will only be feasible if the condition for an epidemic threshold is satisfied:

$$p_c < p < 1. \quad (12)$$

If instead $p_c > 1$, then our system will be more social-like. As we will show below, an initial critical mass will be needed for spreading to take off, if any spreading is possible at all.

To make the epidemic threshold criterion for generalized contagion intuitive, we can combine Eqs. (11) and (12) to form the condition:

$$(p(T)) \cdot P_1^{(\text{inf})} > 1. \quad (13)$$

For a small seed to take off, the interpretation of Eq. (13) tracks as follows. Consider one infected individual at $t = 0$ with a one off dose in their memory exceeding their threshold. They will randomly interact with $T$ different uninfected individuals before they themselves recover. The expected number of exposures they will produce in this time is $p(T)$, the first term in Eq. (13). Because the seed set of infectives is infinitesimally small, each susceptible individual interacted with by an infective will receive at most one dose. And this dose will infect them with probability $P_1^{(\text{inf})}$, the second term in Eq. (13). Thus, $p(T) \cdot P_1^{(\text{inf})}$ is the expected number of new infectives due to one infective, equivalent to the reproduction number $R_0$ of the SIR model. In short, Eq. (13) is the statement that one infective begets at least one new infective, leading to an initial exponential growth of the contagion.

We now need to take some more care as the epidemic threshold for generalized contagion is not as simple as that of SIR contagion. If $p_c < 1$, we must consider whether the transition is continuous or discontinuous. As we saw with the example in Fig. 2, it is always the former for the SIR model.

If the transition is continuous, then when $p = p_c$, a small seed at will not grow, whereas when the transition is discontinuous, spreading will take off rapidly.

To test the phase transition’s continuity, we expand Eq. (8) to second order:

$$\phi_{t+1} \approx (p \phi_{t}) \sum_{T=1}^{T_{\text{max}}} P_T^{(\text{mem})} T P_1^{(\text{inf})} +$$

$$(p \phi_{t})^2 \sum_{T=1}^{T_{\text{max}}} P_T^{(\text{mem})} T (T - 1) \left[ \frac{1}{2} P_2^{(\text{inf})} - P_1^{(\text{inf})} \right]$$

$$= (p \phi_{t}) (T) P_1^{(\text{inf})} +$$

$$(p \phi_{t})^2 (T (T - 1)) \left[ \frac{1}{2} P_2^{(\text{inf})} - P_1^{(\text{inf})} \right]. \quad (14)$$
Setting $p = p_c$, Eq. (11), we have:

$$
\phi_{t+1} \simeq \phi_t + (\phi_t)^2 \frac{1}{(T-1)} \left[ \frac{1}{2} \delta_2(\inf) - \delta_1(\inf) \right].
$$

A discontinuous phase transition is apparent if the fraction infected $\phi_t$ grows and this evidently occurs if right hand side of Eq. (15) is positive, meaning:

$$
P_2^{\text{(inf)}} < 2P_1^{\text{(inf)}} : \text{continuous},
$$

$$
P_2^{\text{(inf)}} > 2P_1^{\text{(inf)}} : \text{discontinuous}.
$$

We see that the kind of contagion behavior we observe with social phenomena, that repeated doses combine superlinearly $P_2^{\text{(inf)}} > 2P_1^{\text{(inf)}}$, corresponds with explosive spreading of a small seed at the critical point. Discontinuous phase transitions are phase transitions of surprise— as we increase the exposure probability $p$ starting well below $p_c$, we see no spreading until we reach $p_c$ (or just below depending on $\phi_0$) when the growth will both be sudden and potentially leading to a large final fraction of infection. If repeated doses combine sublinearly, $P_2^{\text{(inf)}} < 2P_1^{\text{(inf)}}$, then the final fraction of infections will grow continuously from 0 as we move past $p_c$. Now, this is for the special case of a pure SIS model and as we later note, the criterion for a vanishing critical mass model, $P_2^{\text{(inf)}} > 2P_1^{\text{(inf)}}$, does not remain so simple as we move to more complicated models. So, while we can observe that a sufficiently nonlinear interaction in doses leads to non-epidemic threshold model, we arguably should not have been able to intuit the simple inequality $P_2^{\text{(inf)}} > 2P_1^{\text{(inf)}}$ as being the salient test.

We can now assert that the generalized contagion model produces three distinct universality classes with respect to spreading behavior from a small seed. These are:

- **Epidemic Threshold Class:**
  
  Criteria:
  
  1. $p_c = 1/((T)P_1^{\text{(inf)}}) < 1$.
  2. $P_1^{\text{(inf)}} > P_2^{\text{(inf)}}/2$.

- **Vanishing Critical Mass:**
  
  Criteria:
  
  1. $p_c = 1/((T)P_1^{\text{(inf)}}) < 1$.
  2. $P_1^{\text{(inf)}} < P_2^{\text{(inf)}}/2$.

- **Pure Critical Mass:**
  
  Criteria:
  
  1. $p_c = 1/((T)P_1^{\text{(inf)}})$.
  2. Eq. (9) is solvable with solutions $\phi^*(p) \in [0,1]$.

In Fig. 5, we show results from numerically solving Eq. (9) for three example dose distributions and $T = 20$ set uniformly, (see caption for details; adapted from Fig. 9 in [2]).

The three panels correspond in order to the three universality classes. We emphasize that the universality classes we find here relate to the kind of critical point
present in the system for $\phi^* = 0$, if such a critical point exists. The details of these systems are unimportant as many threshold and dose distributions give same $P_k^{(inf)}$. All solid blue curves indicate stable fixed points and dashed red curves unstable fixed points.

For the epidemic threshold in Fig. 5A, we see a continuous phase transition occurring at $p_c = 1/4$. Small seeds for $p$ above $p_c$ will grow but be constrained.

In Fig. 5B, the Vanishing Critical Mass class also shows a epidemic threshold but now the phase transition is discontinuous. Tuning the system from below to above $p_c = 2/3$, a small seed moves from ineffectual to suddenly producing successful global spreading to, roughly, half of the population.

The fixed point curves for the Critical Mass model in Fig. 5C show the resilience of this third class to small seeds initiating spreading events. Only if the initial seed is above the dashed red curves of unstable fixed points, will the final extent of spreading be non-zero (this statement is true for all three classes).

For uniform memory length $T_*$, the full linearization near $p$ has the form [2]:

$$
\phi^* \simeq \frac{C_1}{C_2p_2}(p-p_c) = \frac{T_2^2p_3^3}{(T_*-1)(P_1-P_2/2)}(p-p_c),
$$

where from the denominator we can again see that $P_1 - P_2/2 = 0$ locates the transition between Epidemic Threshold models and Vanishing Critical Mass models.

Moving away from systems behavior for small seeds, in all three examples, we see that the threshold distributions are of enough variability to produce non-trivial fixed point curves. Further, both the Epidemic Threshold and Vanishing Critical Mass examples also show that hysteresis dynamics (with respect to $p$) are available for Generalized Contagion systems.

If we relax the recovery probability $r$ below 1 and/or elevate the immune state transition probability $\rho$ above 0, then we see the same three universality classes will still emerge. The conditions for the three classes will become more complex [2]. The appealing form of the test separating Epidemic Threshold and Vanishing Critical Mass models, $P_2^{(inf)} < 2P_1^{(inf)}$, will no longer be quite so simple. Analytic results are possible for $r < 1$ and $\rho = 0$ [2] while systems with $\rho > 0$ have not yielded, at least to our knowledge, to exact treatments.

VI. CONCLUDING REMARKS

We developed generalized contagion to demonstrate that a single mechanism could be shown to produce both disease-like and social-like spreading behavior. The observation that memory is a natural aspect of real-world spreading phenomena proved to be the binding agent.

The three universal classes of contagion processes pertain to the spectrum of random-mixing models and their dynamics in the fundamental initial condition of an infinitesimally small seed. We see that dramatic changes in behavior are possible, particularly in the Vanishing Critical Mass class.

Generalized contagion is also another example of a model where the vulnerable or gullible population may be more important than a small group of super-spreaders or influencers [20].

Two avenues for changing dynamics are clear. One would be to change the model itself through adjusting its parameters: memory, recovery rates, and the fraction of individuals vulnerable to 1 or 2 doses. ($T, r, \rho, P_1^{(inf)}$, and $P_2^{(inf)}$). Given a model with fixed parameters, changing the system’s behavior would be possible by changing the probability of exposure ($p$) and/or the initial fraction infected ($\phi_0$).

We hope that this overview of generalized contagion serves as both an introduction to the model itself and an inspiration for the many possible adjacent areas in contagion dynamics available for development. Generalized contagion on social-like networks more complicated than random networks would be one such path. While perhaps this work would be resilient to simple analysis, simulations could prove illuminating.

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