Different universality classes of epidemiological models

Gabriel Toneatti Vercelli
Microbiology Program, Massachusetts Institute of Technology, Cambridge, MA, 02139, USA
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The current coronavirus pandemic has been a constant part of everyone's lives for the previous year or so. However, why did Covid-19, out of all diseases, have such a major impact globally? What turns an epidemic into a pandemic? That is the question we answer here with a review on different epidemic models connecting them to percolation theory. We start with a simple SIS model in which people that get the disease, after recovered, can get it again. The flu and other endemic diseases are well described by these types of models. We then move to the most well-know epidemic model, the SIR, in which people become immune to the disease after infection. The last model we go over is the SWIR model, introducing the possibility of people becoming weakened by an initial exposure to the disease, but not infectious. We show that these three models belong to different universality classes by a mean-field theory approach. We also define thresholds separating different phases of these systems and calculate a few of the mean-field critical exponents. Possible applications and experimental avenues to continue this research are discussed in the end.

I Introduction

Having experienced a pandemic, most people nowadays have at least heard of the concept of herd immunity threshold. This threshold — defined as the minimum number of people that must become immune to a disease to stop its propagation — marks the point at which a disease is converted from a pandemic back into an epidemic, with only small local effects. It may come as a surprise, however, that this is not a soft threshold, like IC_{50}'s, half-lives or other biological metrics. It marks instead a fundamental change in the spreading behavior of the disease. If the number of immunized people is above the threshold, the probability of the disease being a pandemic is 0, while below the threshold the probability increases continuously from 0 following a power law. A question one could ask now is: why such a threshold even exists in the first place? Why doesn't the probability of transitioning from an epidemic to a pandemic decrease smoothly with the percentage of immune people?

To answer this question, one can convert classic, well-established epidemic models, like the SIR system of differential equations, into models of percolation. In this review, we will go over this conversion and show how different epidemic descriptions can lead to different universality classes inside percolation problems. For all the models presented, we will assume an underlying spatial structure of a square d-dimensional lattice.

II Epidemic models

A Simple epidemic process (SEP)

A simple epidemic model can be created by considering people positioned in each square of a lattice. The people don't move, but can interact with their nearest-neighbors. This way, when someone is infected by a disease, the infection has a chance k of spreading locally. In this simple model, we also assume that all infected people overcome the infection at a fixed rate λ and then become susceptible again (SIS model, [1]). This system is then described by the reactions

$$\begin{aligned} S(\boldsymbol{n}) + I(\boldsymbol{m}) &\xrightarrow{k} I(\boldsymbol{n}) + I(\boldsymbol{m}) \\ I(\boldsymbol{n}) &\xrightarrow{\lambda} S(\boldsymbol{n}) \end{aligned} \tag{1}$$

Where S and I represent susceptible and infected, respectively. In addition, $\boldsymbol{n}$ represents a lattice site and $\boldsymbol{m}$ one of its neighbors.

B General epidemic process (GEP)

A more general epidemic process assumes that after getting the disease people either die or become immune (SIR model, [2]). Both cases receive an equal mathematical treatment because in both situations the person is being removed from the pool of susceptible individuals. A separation between death and immunity could be achieved by considering death/immunization rates, but this separation is irrelevant for understanding the spread of the disease. The reactions describing this model are

$$\begin{aligned} S(\boldsymbol{n}) + I(\boldsymbol{m}) &\xrightarrow{k} I(\boldsymbol{n}) + I(\boldsymbol{m}) \\ I(\boldsymbol{n}) &\xrightarrow{\lambda} R(\boldsymbol{n}) \end{aligned} \tag{2}$$

Where R represents recovered and lambda now is interpreted as the recovery rate.

C Generalized GEP (GGEP)

A further generalization of the previous model includes the possibility of susceptible individuals not becoming infected from a first exposure to the disease, but rather becoming weak. Weak individuals are then more susceptible to acquiring the disease, but are still not infected and do not spread. The reactions defining such a system are

$$\begin{aligned} S(\boldsymbol{n}) + I(\boldsymbol{m}) &\xrightarrow{k} I(\boldsymbol{n}) + I(\boldsymbol{m}) \\ S(\boldsymbol{n}) + I(\boldsymbol{m}) &\xrightarrow{\mu} W(\boldsymbol{n}) + I(\boldsymbol{m}) \\ W(\boldsymbol{n}) + I(\boldsymbol{m}) &\xrightarrow{\nu} I(\boldsymbol{n}) + I(\boldsymbol{m}) \\ I(\boldsymbol{n}) &\xrightarrow{\lambda} R(\boldsymbol{n}) \end{aligned} \tag{3}$$

Where W represents weak individuals, μ is the rate at which exposure makes an individual weak, and $\nu > k$ is the susceptibility of the weak individuals to infection [3].

III Mean-field approach

In a mean-field approximation, we average out the fluctuations in the previous stochastic reactions and write them as deterministic differential equations. Our goal then is to find the steady-state solutions to those equations and their stability. We describe the procedure for the SEP model. Rewriting eq. (1) using $\mathcal{N}_n$ to denote the neighborhood of the site $\boldsymbol{n}$, we get

$$\begin{aligned} \dot{S}(\boldsymbol{n},t) &= -kS(\boldsymbol{n},t) \sum_{\boldsymbol{m}\in\mathcal{N}_n} I(\boldsymbol{m},t) + \lambda I(\boldsymbol{n},t) \\ \dot{I}(\boldsymbol{n},t) &= kS(\boldsymbol{n},t) \sum_{\boldsymbol{m}\in\mathcal{N}_n} I(\boldsymbol{m},t) - \lambda I(\boldsymbol{n},t) \end{aligned} \tag{4}$$

We further have the condition that $S + I = 1$ at each site, interpreting S and I as the probability of a site being susceptible or infected, respectively. Using this relation to eliminate S in the equation for I, we arrive at

$$\dot{I}(\boldsymbol{n},t) = k(1 - I(\boldsymbol{n},t)) \sum_{\boldsymbol{m}\in\mathcal{N}_n} I(\boldsymbol{m},t) - \lambda I(\boldsymbol{n},t) \tag{5}$$

Taking the continuum limit of the lattice, we can say that $\sum_{\boldsymbol{m}\in\mathcal{N}_n} I(\boldsymbol{m},t) = a^2\boldsymbol{\nabla}^2 I(\boldsymbol{x},t) + zI(\boldsymbol{x},t)$. This is the generalization of the 4 point approximation of a two-dimensional derivative, where we have used z to denote the connectivity of the lattice and a to denote the characteristic lattice length scale.

Using that limit, then, we arrive at a reaction-diffusion equation of the form

$$\dot{I}(x,t) = D\boldsymbol{\nabla}^2 I - DI\boldsymbol{\nabla}^2 I - \lambda I(g + fI) \tag{6}$$

Where we have defined $D = ka^2$, $g = 1 - zk/\lambda$, and $f = zk/\lambda$. Defining $\theta^{-1} = zk/\lambda$, we see that we can describe the dynamics of homogeneous solution with only one parameter. The important question to ask now is how the system responds to a small localized perturbation in I (an initial infection). At most sites, I will be close to 0, except at the point of infection. We can then approximate the previous equation to a homogeneous solution and understand how that solution at positions away from 0 respond to the small perturbation around the origin. Setting $I(x,t) = I_\infty$, we arrive at solutions

$$I_\infty = 0 \text{ or } -\frac{g}{f} \tag{7}$$

In function of θ, the second solution can be written as $1 - \theta$, which is between 0 and 1 only when $0 < \theta < 1$. A stability analysis shows that $I_\infty = 0$ is stable only for $\theta > 1$, while $I_\infty = 1 - \theta$ is stable only for $\theta < 1$. Together, these results define the critical value of $\theta_c = 1$. For $\theta > \theta_c$, the disease cannot spread and the perturbation dies out quickly by all people becoming susceptible. For $\theta < \theta_c$, but still close to the threshold value, the disease spreads indefinitely with the probability of a site being infected increasing as $I = 1 - \theta = \theta_c - \theta$. The fact that the probability is not 0 nor 1 indicates that the sites continuously alternate between susceptible and infected states in a realization of the system. The system, therefore, never reaches a stable state, but rather maintains dynamical activity indefinitely.

The continuous increase of I at the critical point signals a second order phase transition and, interpreting I as an order parameter, we find the critical exponent $\beta = 1$ for the mean field solution of the SIS model. Testing how individual Fourier modes behave in eq. (1) by setting $I(x,t) = \tilde{I}(q,t)e^{-i\boldsymbol{q}\cdot\boldsymbol{x}}$, one can uncouple lattice points in Fourier space and arrive at a condition for modes $\tilde{I}(\boldsymbol{q},t)$ to converge to a non-trivial value. This condition is

$$q^2 < \frac{\lambda}{D}\left(\frac{\theta_c - \theta}{\theta}\right) \tag{8}$$

This means that for $\theta > \theta_c$, all Fourier frequencies converge to 0, representing the disordered phase. On the other hand, as θ passes through θ_c becoming smaller, low frequency modes start to become non-trivially stable. We can then define a correlation length $\xi^{-1} = \sqrt{\frac{\lambda}{D}\left(\frac{\theta_c-\theta}{\theta}\right)}$ such that all frequencies with $q < \xi^{-1}$ have non-trivial stable solutions. This correlation length can be interpreted as a linear estimate of the size of the region not affected by the infection (the infection cloud). As $\theta \to \theta_c^-$, the linear size of the infection cloud shrinks while the linear length of the susceptible regions diverges. The exponent controlling that divergence is $\nu = 1/2$.

These exponents are exact only when the mean-field approximation is valid, which happens for dimensions

above an upper critical dimension d_u when fluctuations become negligible in the system. Since SIS models belong to the directed percolation (DP) universality class, the upper critical dimension is $d_u = 4$. To calculate corrections to the critical exponents below this dimension, other methods such as RG or exact treatments are needed.

The DP universality class describes percolating systems with a transition between an active phase and an absorbing phase. In the active phase, the dynamical behavior extends indefinitely, like in the previous model for $\theta < \theta_c$ where people keep fluctuating between infected and non infected states. In the absorbing phase, no fluctuations exist, like in the previous model for $\theta > \theta_c$ where the system converges to all people being susceptible, without any stochastic fluctuations.

Similar analyses can be done for the other models focusing on the dynamics of R. We start with the GEP model. Transforming the reaction equations into differential equations is straightforward and the equations can be solved as in [3] using the conservation law $S + I + R = 1$ at each site to arrive at the continuous spatial formulation

$$\dot{R}(\boldsymbol{x}, t) = \lambda(1 - R) - \lambda S(\boldsymbol{x}, 0) e^{-\frac{D}{\lambda}\boldsymbol{\nabla}^2 R - \frac{zk}{\lambda} R} \tag{9}$$

This model always converges to a steady state, simply because in a finite lattice either the infection dies out, or all people become recovered. Because these two options occur at opposite ends of the model parameter space, we expect R to exhibit a phase transition connecting these two regimes for an intermediate parameter value. Away from the initial infection, we expect R to change slowly, so we consider homogeneous solutions to the previous equation. Assuming the initial configuration had $S(\boldsymbol{x}, 0) = 1$ for lattices away from the initial infection, we get the equation of state

$$R = 1 - e^{-\frac{zk}{\lambda} R} \tag{10}$$

Again, naming $\theta^{-1} = zk/\lambda$, we see that the previous equation has a nontrivial stable solution only for $\theta < 1$. For $\theta > 1$, the only stable solution is $R = 0$. The critical point, then can be defined as $\theta_c = 1$. Close to it, R is small and we can approximate the equation as

$$R = \frac{R}{\theta} - \frac{R^2}{2\theta^2} + O(R^3) \tag{11}$$

with solution $R = 2\theta(1-\theta) = 2\theta(\theta_c - \theta)$, for $\theta < \theta_c = 1$ and $R = 0$ for $\theta > \theta_c$. Therefore, interpreting R as an order parameter, we have $\beta = 1$ as one of the critical exponents. Looking at individual Fourier modes like before, we arrive at a condition for $\tilde{R}(\boldsymbol{q}, t)$ to have a stable non-trivial equilibrium

$$q^2 < \frac{\lambda}{D}\left(\frac{\theta_c - \theta}{\theta}\right) \tag{12}$$

This is exactly the same condition as before, so as θ decreases and crosses the threshold, we go from not having any stable modes to having stable modes with small frequencies q. In addition, we can define a correlation length $\xi^{-1} = \sqrt{\frac{\lambda}{D}\left(\frac{\theta_c - \theta}{\theta}\right)}$, analogously controlled by the parameter $\nu = 1/2$. The similarity close to the critical point comes from the fact that eqs. (6) and (9) have the same linear terms, if we expand the exponential in powers of R and $\boldsymbol{\nabla}^2 R$. Importantly, however, they differ for higher order terms, and that changes the value of the order parameter in the ordered phase.

Both exponents calculated are the same for the SIS and the SIR model. Nevertheless, these two models are in different universality classes and for these classes one needs 3 critical exponents to define all the other ones [4]. As explained before, the SIS model is in the DP universality class, but the SIR model is in the isotropic percolation (IP) universality class. The main qualitative difference between IP and DP is that IP connects two absorbing phases — all people being susceptible, and all people being recovered — as opposed to an absorbing to an active phase like DP. This difference manifests itself in many dynamical exponents not calculated here, but these classes also have different upper critical dimensions. The upper critical dimension for IP is $d_u = 6$, rather than the more usual value of $d_u = 4$ found for DP and many other universality classes [5].

The last model we study here is the GGEP model. Similarly to before, we can transform the reactions defined into deterministic differential equations and take the continuum limit of space. Using, then, that $S + W + I + R = 1$ at each lattice site we arrive at the equation

$$\begin{aligned}\dot{R}(\boldsymbol{x}, t) = {} & \lambda(1 - R) \\ & - \lambda S(\boldsymbol{x}, 0)\left[\frac{\rho - k}{\rho - \nu} e^{-\frac{\nu}{\lambda}(a^2\boldsymbol{\nabla}^2 R + zR)}\right. \\ & \left. + \frac{k - \nu}{\rho - \nu} e^{-\frac{\rho}{\lambda}(a^2\boldsymbol{\nabla}^2 R + zR)}\right]\end{aligned} \tag{13}$$

where we have defined $\rho = k + \mu$.

Like before, we consider an initial condition with most $S(\boldsymbol{x}, 0) = 1$ and a small infection close to the origin. We then look at points for which $|x| >> 1$ so that R is slowly spatially variant and we can drop the gradient terms. Looking, then, for steady state solutions we arrive at the equation of state

$$R = 1 - e^{-\frac{\nu}{\lambda} zR} + \frac{\nu - k}{\rho - \nu}\left[e^{-\frac{\rho}{\lambda} zR} - e^{-\frac{\nu}{\lambda} zR}\right] \coloneqq f(R) \tag{14}$$

Clearly, $R = 0$ is always a solution for this equation. From eq. (13), we see a solution R^* is stable when $f'(R^*) < 1$. Since $f'(0) = \frac{zk}{\lambda}$, the order parameter $\theta^{-1} = \frac{zk}{\lambda}$ again has a threshold value at $\theta_c = 1$.

At this threshold, a second order transition from the stable solution $R = 0$ to a non-zero stable solution happens. For $\theta < \theta_c$, $R = 0$ is unstable and there is at least one non-zero stable solution because $f'(0) > 1$ but $f(R) \to 1$ as $R \to \infty$.

More than one stable solution is possible, however, and a necessary condition is $f''(R) = 0$ for some R between stable states. We know that $f''(R) < 0$ for $R >> 1$, $f''(0) = \frac{z^2}{\lambda^2}(\rho\nu - k(\rho + \nu))$, and it is easy to show that for $R_2 > R_1$

$$f''(R_2) \leq \frac{z^2}{\lambda^2} f''(R_1) \exp\Big(-\frac{z}{\lambda}(R_2 - R_1)\max(\rho, \nu)\Big) \tag{15}$$

Therefore, for $R_1 = 0$ we get the condition

$$\frac{1}{k} > \frac{1}{\rho} + \frac{1}{\nu} \tag{16}$$

and this is necessary and sufficient for $f''(R)$ to have a 0. Note that by taking R_2 to be the position of the first zero, we show that there can only be one zero in $f''(R)$ and consequently at most 2 stable states. If $\theta < \theta_c$ and eq. (16) is valid, the point R^* in which $f''(R^*) = 0$ must be before the first stable state, and therefore we only have one stable state in these conditions. For $\theta > \theta_c$ and eq. (16), a new non-zero stable equilibrium appears and this represents the possibility of a discontinuous transition when θ passes θ_c. The tricritical line where all these three stable states exist is determined by

$$\frac{1}{k} = \frac{1}{\rho} + \frac{1}{\nu} = \frac{z}{\lambda} \tag{17}$$

in the $\rho - \nu - k$ phase space.

This is a strikingly different behavior from the ones found before and the universality class for this model is the tricritical dynamic isotropic percolation (TdIP) with an upper critical dimension of $d_u = 5$ [3].

IV Conclusions

Going back to the thought we started with, this work shows how different microscopic spreading properties can give rise to completely different global properties of epidemics. Understanding with which class one is dealing with is essential for designing suitable public policies and responding readily to crises.

Some of the nomenclature we used here has other names in epidemiology. The order parameter, θ, is most often called the reproductive number, R. And the immunity threshold, the concept we started with, can be thought of as an intervention tuning the parameter z because immunizing people in the lattice decreases its connectivity. With simple calculations its possible to show that for a fraction larger than $1 - \frac{1}{\theta}$ of the people vaccinated, the effective order parameter becomes larger than one, preventing continuous spread of the disease. Therefore, the herd immunity threshold for a disease is defined as $T = 1 - \frac{1}{\theta}$.

Interesting experimental directions that could be taken based on these theoretical predictions is to verify that epidemic do, indeed, belong to these universality classes by estimating their critical exponents. One could also explore the idea of self-organized criticality (SOC), which poses that many natural systems are tuned to criticality, by evolution or other means. In the case of pandemics, that would entail not only validating critical exponents, but looking at the distribution of different diseases in the parameter spaces available to biological systems. A clustering around critical lines and critical thresholds would support the idea of SOC.

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