

The SIR dynamic model of infectious disease transmission and its analogy with chemical kinetics

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Abstract

The classic Susceptible-Infectious-Recovered (SIR) mathematical model of the dynamics of infectious disease transmission resembles a dynamic model of a batch reactor carrying out an autocatalytic reaction with catalyst deactivation. By making this analogy between disease transmission and chemical reactions, chemists and chemical engineers can peer into dynamic models of infectious disease transmission used to forecast epidemics and assess mitigation strategies.

Mathematical models of the dynamics of infectious disease transmission [1, 2] are useful for forecasting epidemics, assessing intervention strategies, and inferring properties of diseases.

In compartmental epidemic models [3], each member of the population is categorized based on their disease status and (possibly) attributes. The dynamics of disease transmission are then typically modeled with a set of differential equations that describes the flow of individuals to and from the compartments as the population mixes, the disease is spread/contracted, and infected individuals progress through the stages of the disease. Differential equations are a natural choice because we can make reasonable assumptions about the *rates* at which people are infected and recover. In this article, we highlight the analogy between compartmental epidemic models and dynamic models of chemical reactions.

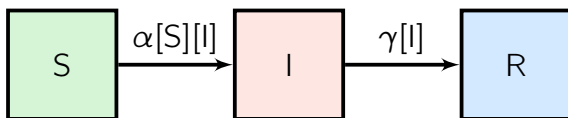


Figure 1: The SIR model. The boxes represent the set of Susceptible, Infectious, and Recovered individuals. The arrows represent flow from one compartment to another and are annotated with flow rates normalized by the population size.

In the classic SIR model of an epidemic [2, 4–6], each member of the population belongs to one of three compartments: Susceptible, Infectious, or Recovered. Fig. 1 depicts the flow of individuals through compartments, assuming that the disease confers immunity to re-infection after recovery.

Susceptible folks can contract the disease if they come into contact with an infectious individual.

Once infected, they move into the infectious compartment, assuming zero delay between infection and the ability to transmit the disease. This is analogous to an irreversible autocatalytic chemical reaction [7, 8] between a reactant, S, and catalyst, I:



Infectious individuals eventually recover from the disease, entering the recovered compartment, and then cannot transmit the disease or contract it again. This is analogous to a reaction where the catalyst, I, irreversibly degrades or converts to a deactivated form R:



So, the SIR model of an epidemic is analogous to an autocatalytic reaction with catalyst deactivation. An infectious individual (the catalyst, I) (i) converts susceptibles (the reactant, S) into more infectious individuals (more catalyst) and (ii) recovers (deactivates) with time.

Mathematically, the SIR model [4–6] is equivalent to a dynamic model of a well-mixed, isothermal batch reactor carrying out the two homogeneous, elementary reactions {1} and {2}. Let [S], [I], and [R] be the fraction of the population in the susceptible, infectious, and recovered compartments, respectively.

The incidence rate. Assuming their spatial mixing is uniform [9,10], we invoke the law of mass action to model the rate at which susceptible and infectious individuals “react”, as in a bimolecular reaction. The incidence rate of the disease, i.e. the rate at which new infections occur [11], is then $\alpha[S][I]$ (normalized by population size). A symmetric function of [S] and [I], intuitively, the incidence rate doubles if [I] ([S]) doubles while [S] ([I]) is fixed. The second-order transmission rate constant $\alpha > 0$ encapsulates both the degree of mixing between susceptible and infectious individuals (frequency of contacts) and the transmissibility of the disease (the probability of transmission conditioned upon contact) [11, 12].

The recovery rate. We posit that infectious individuals “decay” (recover) with first-order kinetics, i.e., with rate $\gamma[I]$ (normalized by population size). The inverse of the first-order recovery rate constant $\gamma > 0$ is the average time period that an infected individual is infectious¹ [13].

Immigration, emigration, births, and deaths. As in a closed batch reactor, we neglect immigration and emigration. Moreover, we take births and deaths from causes other than the disease to be negligible over the time scale of the epidemic. Consequently, $[S](t) + [I](t) + [R](t) = 1, \forall t \geq 0$. Deaths caused by the disease are counted as $I \longrightarrow R$ transitions, assuming the deceased cannot transmit the disease. The R category is often called the Removed category [14], emphasizing its inclusion of disease-induced deaths.

With the assumptions above (see Fig. 1 for flows), we arrive at the following set of nonlinear, coupled

¹Specifically, the probabilistic implication of first-order decay for a single infected individual is that their time period of infectiousness is an exponentially distributed random variable with mean γ^{-1} [3].

differential equations that comprise the SIR dynamic model of infectious disease transmission:

$$\frac{d[S]}{dt} = -\alpha[S][I] \quad (1)$$

$$\frac{d[I]}{dt} = \alpha[S][I] - \gamma[I] \quad (2)$$

$$\frac{d[R]}{dt} = \gamma[I]. \quad (3)$$

The only two parameters in the SIR model are the transmission and recovery rate constants, α and γ , respectively. While γ could be estimated independently from studies on the duration of infectiousness, α could be identified by fitting differential eqns. 1-3 to epidemic time series data (case counts) [10, 11], much like identifying a reaction rate constant from concentration time series [15].

In the SIR model, what happens if we introduce a *single* infectious individual into a population of entirely susceptible individuals? This is akin to introducing our deactivating auto-catalyst into a batch of reactant. Intuitively, if the catalyst has a sufficiently high activity and/or remains active long enough, it will initiate a reaction (an epidemic). To the contrary, if the catalyst has a low activity and/or quickly deactivates, it will not initiate a reaction. The activity and longevity of the catalyst are embedded in α and γ , respectively. Indeed, the qualitative outcome of introducing an infectious individual into a population of susceptible folks depends on the (dimensionless) basic reproduction number $\mathcal{R}_0 := \alpha/\gamma$, a property of both the disease and the population [16]. See Appendix A. If $\mathcal{R}_0 < 1$, the infectious recover quickly, the disease is not easily transmitted, and/or the mixing of susceptibles and infectious is not vigorous. Consequently, an epidemic will not ensue; $[I](t)$ decreases monotonically. If $\mathcal{R}_0 > 1$, the infectious are infectious for a long period of time, the disease is easily transmitted, and/or the mixing of susceptibles and infectious is vigorous. Consequently, the single infectious individual propagates the disease and starts an epidemic; $[I](t)$ grows, approximately and only initially, exponentially (see Appendix B). Explaining this threshold behavior and the terminology “reproduction number”, \mathcal{R}_0 is the expected number of folks (directly) infected by this infectious individual introduced into the all-susceptible population over the course of their infectiousness [6] (see Appendix A.).

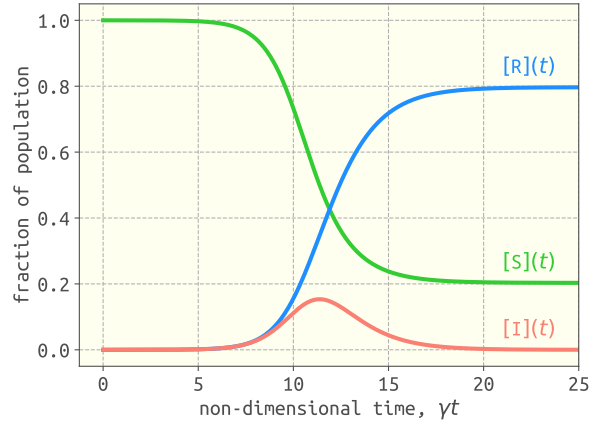


Figure 2: Numerical approximation of the solution to the SIR model in eqns. 1-3 for $\mathcal{R}_0 = 2$. Initial conditions: $[I](t = 0) = 1/100000$, $[S](t = 0) = 99999/100000$, and $[R](t = 0) = 0$.

Fig. 2 shows a simulation of SIR model dynamics for $\mathcal{R}_0 = 2$ (see Appendix C for code). In the initial stage of the epidemic, the number of infectious individuals grows approximately exponentially with growth rate $\gamma(\mathcal{R}_0 - 1)$ (see Appendix B). As the disease propagates, the concentration of susceptible individuals decreases, eventually causing the incidence rate of the disease to diminish. Interestingly,

in conjunction with the infectious folks recovering, this causes the epidemic to die out before the entire population is infected. That is, an SIR model epidemic (case $\mathcal{R}_0 > 1$) does not result in *every* susceptible member of the population being infected, even after an infinite amount of time (see Fig. 2). From a chemical engineer's standpoint, the reaction begins to die out ($\frac{d[I]}{dt} < 0$) when the concentration of the reactant, $[S]$, becomes so low that any given catalyst particle, I , is expected to deactivate before it can convert a reactant molecule, S , into another catalyst particle to replace itself. i.e., an SIR epidemic dies out not because the population is depleted of susceptible folks, but rather because it is depleted of infectious folks [5,10]. The fraction of the reactant consumed (fraction of susceptibles infected) at the end of the reaction (epidemic) depends on both the activity and longevity of the catalyst (frequency of S - I contacts, transmissibility of the disease, time period of infectiousness; all of which are embedded in \mathcal{R}_0). By the end of the epidemic, the fraction of the population that will not have been infected, $[S]_\infty := \lim_{t \rightarrow \infty} [S](t)$, is related to the basic reproduction number (see Appendix D):

$$\log[S]_\infty = ([S]_\infty - 1)\mathcal{R}_0. \quad (4)$$

Fig. 3 shows $[S]_\infty$ as a function of the basic reproduction number; as \mathcal{R}_0 increases from one, more of the population will have been infected over the course of the epidemic.

To summarize, the basic reproduction number \mathcal{R}_0 is defined in the context of introducing a single infectious individual into an entirely susceptible population [12], and, in the SIR model, it determines whether or not an epidemic will ensue, the initial exponential growth rate of the number of infectious individuals (in non-dimensional time, γt), and the fraction of the population that will have been infected over the course of the epidemic.

The mechanism by which a vaccination program provides herd immunity to a population is mathematically similar to the time in Fig. 2 when the epidemic begins to die off: owing to a sufficiently small concentration of susceptible folks (achieved by vaccination or, as is the case in Fig. 2, by a fraction of the population having been infected, recovered, and conferred immunity), the infectious recover faster than they spread the disease to the susceptible folks to replace themselves [17]. A simple way to model vaccination in the SIR model is to allow flow in Fig. 1 from the S category directly to the R category when a vaccine that confers complete immunity is administered to susceptible folks [11,18,19]. A chemical engineering view of herd immunity is the reduction of the concentration of the reactant, $[S]$, achieved by either vaccination or by recovery from infection, so that a catalyst particle, I , fed to the reactor is expected to deactivate before it encounters an S particle and auto-catalyzes a reaction $S \rightarrow I$.

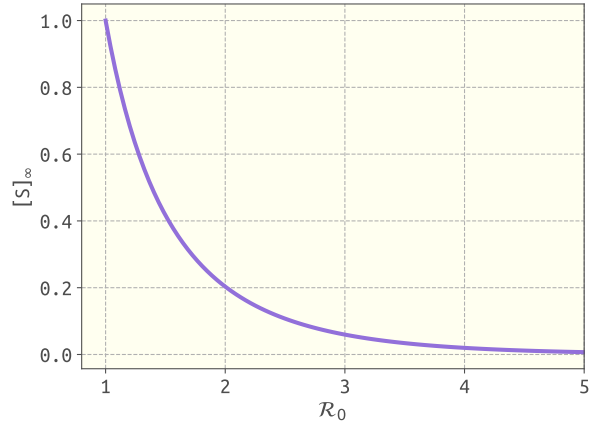


Figure 3: The fraction of the population that remains susceptible (never infected) at the end of the SIR epidemic, $[S]_\infty$, as a function of the basic reproduction number \mathcal{R}_0 , computed via eqn. 4.

The SIR model is a very simple compartmental model, but we can extend it to account for other factors that influence disease transmission by introducing:

(i) *additional compartments*. To model the latent period of a disease, the SEIR model contains an E compartment for Exposed individuals that have been infected but are not yet infectious [3, 20]. To model intervention strategies, a Q compartment for Quarantined infectious individuals accounts for the reduced frequency of contacts of susceptible folks with infectious individuals that are quarantined [3]. To distinguish between Asymptomatic and symptomatic infectious individuals [21], which may have different recovery rates, frequencies of contacts with susceptible folks, and transmissibilities, we can introduce an A compartment. To account for different mixing patterns, infectiousness, and susceptibility among different age groups [3], age-structured compartmental models partition the S and I compartments into age groups.

(ii) *time-varying parameters*. For example, if we allow the transmission rate constant α to vary with time, we can model (a) members of the population changing/adapting their behavior, e.g., reducing their frequency of social interactions and practicing social distancing [22, 23] and (b) seasonality of an infectious disease [24, 25].

(iii) *stochasticity*. As opposed to the deterministic differential eqns. 1-3, we can introduce randomness into the SIR model to account for the stochastic and uncertain nature of human interaction and disease transmission [26, 27]. Stochastic epidemic models aim to describe the probabilistic distribution of outcomes, e.g., the distribution of $[S]_{\infty}$ [26, 27]. Stochasticity can be particularly important for small populations and in the early stage of an epidemic when there are small numbers of infectious individuals [27]. Analogously, stochastic models of chemical reaction dynamics are necessary when the reactants are not abundant, such as in a biological cell [28, 29].

(iv) *spatial heterogeneity*. Finally, we can model spatial heterogeneity in an epidemic in a discrete [30, 31] or continuous [11] manner and allow for more detail in the interactions/mixing within a population or between populations [32, 33]. Modeling the spatial movement of susceptible and infectious individuals as a diffusive process results in reaction-diffusion equations, also familiar to chemists and chemical engineers [11]. Compartmental, metapopulation epidemic models with travel between spatially segregated regions resemble models of multiple batch reactors connected with pipes that allow flow between them [30, 34].

In conclusion, by making an analogy between disease transmission and chemical reactions, chemists and chemical engineers can peer into dynamic models of infectious disease transmission. Moreover, this is a nice example of how concepts in one field can aid understanding and generate insights in another field.

the appendix

For sections A, B, C, and D below, consider introducing one infectious individual into a population comprised of $N - 1$ susceptible folks. The initial conditions here are:

$$[S](t = 0) = \frac{N - 1}{N} \quad (5)$$

$$[I](t = 0) = 1/N \quad (6)$$

$$[R](t = 0) = 0 \quad (7)$$

If N is large, $[S](t = 0) \approx 1$.

A the basic reproduction number, \mathcal{R}_0

Qualitatively, if $\frac{d[I]}{dt}|_{t=0} > 0$, the disease will propagate, and an epidemic will ensue; if $\frac{d[I]}{dt}|_{t=0} < 0$, the infectious individual will recover before he/she transmits the disease to others [12]. According to eqn. 2 and our initial conditions, under the approximation of large N ,

$$\left. \frac{d[I]}{dt} \right|_{t=0} \approx \frac{1}{N}(\alpha - \gamma). \quad (8)$$

and it is apparent that the number of infectious individuals increases if $\alpha - \gamma > 0$ and decreases if $\alpha - \gamma < 0$. Consequently, the (dimensionless) basic reproduction number $\mathcal{R}_0 := \alpha/\gamma$ determines if an epidemic ensues upon introducing an infectious individual into the population of susceptible folks ($\mathcal{R}_0 > 1$) or not ($\mathcal{R}_0 < 1$). To further interpret \mathcal{R}_0 , eqn. 8 shows that rate of change of the number of infectious individuals ($\frac{d(N[I])}{dt}$) at $t = 0$ is equal to the incidence rate of the disease, i.e. the rate at which new infections occur, which is approximately α , minus the rate of recovery of the infectious individual, γ . Thus, if $\mathcal{R}_0 < 1$, the infectious individual is expected to recover before they can transmit the disease to another person. If $\mathcal{R}_0 > 1$, the infectious individual is expected to transmit the disease before they recover, thereby initiating an epidemic.

The incidence rate of the disease (number of new infections per time) according to eqn. 1 is $N\alpha[S][I]$. Via the initial condition in eqn. 6 and our approximation of eqn. 5 as $[S](t = 0) \approx 1$, the incidence rate of the disease, caused by this single infectious individual, is approximately α new infections per time over the course of their infectiousness. Since γ^{-1} is the mean infectious period, implied by eqn. 2 [3], the expected number of new infections directly produced by this single infectious individual over the course of their infectiousness is $\alpha\gamma^{-1} = \mathcal{R}_0$. Thus, the basic reproduction number \mathcal{R}_0 is the expected number of infections produced directly by the single infectious individual introduced into the (completely susceptible) population over the time span of their infectious period.

B approximately, $[I]$ grows exponentially in the initial stage of an epidemic

If $\mathcal{R}_0 > 1$, in the initial stage of the epidemic, the number of infectious individuals grows approximately exponentially with growth rate $\gamma(\mathcal{R}_0 - 1)$ [3]. In the initial stage of the epidemic where the approximation $[S] \approx 1$ holds, via eqn. 2:

$$\frac{d[I]}{dt} \approx (\alpha - \gamma)[I]. \quad (9)$$

Consequently, in the initial stage of the epidemic, we see exponential growth:

$$[I](t) \approx [I](t=0)e^{\gamma(\mathcal{R}_0-1)t}. \quad (10)$$

Since eqn. 10 is also a valid approximation for $\mathcal{R}_0 < 1$, it reinforces that introducing an infectious individual into a completely susceptible population will not result in an epidemic if $\mathcal{R}_0 < 1$, since $[I](t)$ will show exponential decay if $\mathcal{R}_0 < 1$. Fig. 4 shows $[I](t)$ from the simulation in Fig. 2 ($\mathcal{R}_0 = 2$) along with the exponential growth approximation in eqn. 10.

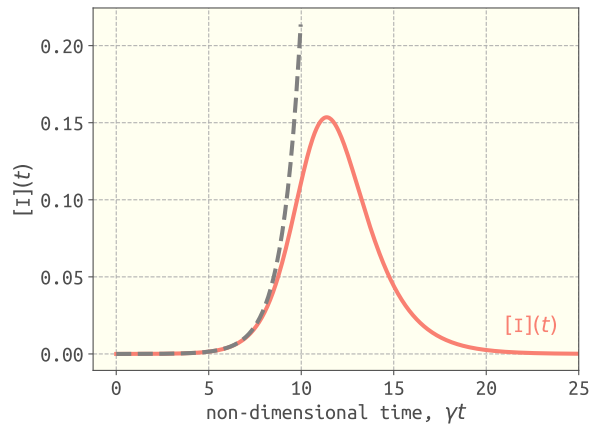


Figure 4: The $[I](t)$ curve from Fig. 2 along with eqn. 10 (dashed line) to show that $[I](t)$ exhibits, approximately, exponential growth in the early stage of the epidemic.

C simulating the SIR model in Julia

Code in the Julia language to numerically approximate the solution to the SIR model, via the DifferentialEquations.jl [35] package, is below.

```
R0 = 2.0      # basic reproduction number
N = 100000    # population size

# right-hand side of the ODE, viewed as:
# du/dt = f = f(u, p, t)
# where u := [S, I, R]
function update_f!(f, u, p, t)
    # for clarity, unpack vector u
    s = u[1]
    i = u[2]
    r = u[3]
    # update f
    f[1] = -R0 * s * i
    f[2] = R0 * s * i - i
    f[3] = i
end

# initial condition
u0 = [(N-1)/N; 1/N; 0.0]

# define the ODE problem
time_span = (0.0, 25.0)
prob = ODEProblem(update_f!, u0, time_span)
```

```
# numerically solve ODE
```

```
sol = solve(prob)
```

```
# obtain numerical approximation to the solution at e.g. t = 10
```

```
sol(10.0)
```

D the fraction of the population that were never infected after the epidemic ends, $[S]_\infty$

After the epidemic ends (as $t \rightarrow \infty$), how many susceptibles will be left? Our derivation follows Ref. [36], but see Ref. [3] for a different derivation.

Adding differential eqns. 1 and 2:

$$\frac{d[S]}{dt} + \frac{d[I]}{dt} = -\gamma[I]. \quad (11)$$

Integrating this from $t = 0$ to $t \rightarrow \infty$:

$$\int_0^\infty \left(\frac{d[S]}{dt} + \frac{d[I]}{dt} \right) dt = - \int_0^\infty \gamma[I] dt. \quad (12)$$

The initial and final values of $[S]$ and $[I]$ appear from the left-hand side. The right-hand side can be written in terms of $[S]$ by solving differential eqn. 1 for $[I]$:

$$\lim_{t \rightarrow \infty} ([S](t) + [I](t)) - ([S](0) + [I](0)) = \frac{\gamma}{\alpha} \int_0^\infty \frac{1}{[S]} \frac{d[S]}{dt} dt. \quad (13)$$

$$= \frac{1}{\mathcal{R}_0} \log[S] \Big|_{t=0}^\infty \quad (14)$$

At the end of the epidemic, all infectious will have recovered, so $[I](t \rightarrow \infty) = 0$. Considering that all of the population was initially susceptible or infectious ($[S](0) + [I](0) = 1$) and approximating $[S](0) \approx 1$, we arrive at:

$$[S]_\infty - 1 = \frac{1}{\mathcal{R}_0} \log[S]_\infty, \quad (15)$$

which is equivalent to eqn. 4.

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