“My teacher sneezed on Monday: SIR Model”

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1. My teacher sneezed on Monday

It was my typical 2024 fall Monday at school. The teacher entered the classroom and sneezed slightly. On Tuesday, 16 students fell ill and stayed at home and on Wednesday 7 more (including me). On Friday we had online lessons, class was closed for quarantine. Do you believe me?

Such mini-epidemics happen everywhere, from my class to global pandemics like COVID-19. After all, epidemics are not an accident. The SIR model was introduced in 1927 by Scottish biochemist William Ogilvy Kermack and physician Anderson Gray McKendrick. They created the modern mathematical framework for infectious disease model which answered a long-standing me question: “Why do waves of disease explode but then fade out?”

2. SIR Model

The SIR model is a simple mathematical model of how infectious diseases spread. It is divided into three groups:

S-Susceptible: healthy people who may be infected. My 30 classmates.

I-Infected: those who are sick and able to transmit an infection. Only 1 teacher.

R-Recovered: those who have recovered and become immune. No one in class.

These variables (S, I, R) represent the number of people in each compartment at a particular time. Imagine that we do the photos of the class every day. That’s what these “compartments” are. To capture the fact that the numbers of susceptible S, infected I, and recovered R individuals can change over time (while the total population size stays constant), we treat them as functions of time t: S(t), I(t), and R(t).

Figure 1: How the disease spread throughout the class

3. Transition Rates

As an epidemic unfolds, the pool of susceptibles $S(t)$ shrinks rapidly: more people become infected $I(t)$ and then recover $R(t)$. Once too few susceptibles remain, the disease fizzles out, unable to spark another major wave until fresh susceptibles appear — say, through newborns. Individuals typically journey through these three compartments in sequence.

Figure 2: Transition rate graph

Between *S* and *I*, the transition rate is assumed to be

$$\frac{dI}{dt} = \beta \frac{SI}{N}$$

The left side is a rate of growth of infected and the right side is a flow intensity.

N is total population (full class including teacher).

β is the speed of infection where implies contacts per day × probability of infection per contact (the teacher infects ~3 of 6 contacts per day, students infect ~1 of 10 contacts per day~). The probability of the [1]basically concerns as a 3-8%.

$\beta = 15 \times 0.053 \approx 0.8$ (for our class)

But when we express the transition rate as a percent of the population, the growth rate of infected must also become a percentage. The equation becomes

$$\frac{d\left(\frac{S}{N}\right)}{dt} = -\beta\, SI/N^2$$

SI/N^2 is fraction of contacts between sick (I) and healthy (S) from all possible pairs in population. This is also similar to the chemical law of mass action: more "reactants" (S × I) = faster reaction.
There is no difference between equation from S and I on the diagram and that big. However, the last one allows us to work not only with my class, but also with population of a country or world.

Between I and R, the transition rate implies the number of infection individuals and is assumed to be

$$\frac{dR}{dt} = \gamma I$$

γ is the speed of recovery and $\gamma = 1/D$, where D is average duration of the infectious period (each pupil stays infectious for D days).

$\gamma = \frac{1}{2.5} = 0.4$ (for our class)

For running into percentage, it will look like

$$\frac{d(R/N)}{dt} = \gamma(I/N)$$

4. How many people has the teacher infected: reproduction number

Still, back in 2024, my question was about the wave of the epidemic that happened in our class. The answer is the basic reproduction number, R_0, which counts how many new cases one infectious person creates on average in a fully susceptible population. R_0 does not change, it is a constant throughout the epidemic.

$$R_0 = \beta k D \text{ or } R_0 = \frac{\beta}{\gamma}$$

Where k is the number of contacts per day, β is the infection rate, γ is the recovery rate. There are still formulas that are based on post-epidemic data, but these two help predict the replication of the disease.

In our class, $R_0 = \frac{0.8}{0.4} = 2$. It depended on how contagious the teacher's flu is and how often students meet each other.

As more people get infected and recover, the fraction of healthy students (S/N) drops, and the outbreak weakens. This is described by the R_e - effective reproduction number.
R_e changes, it is a non constant throughout the epidemic. We consider how many people get infections from one infected at the current time.

$$R_e = R_0 \cdot \frac{S}{N}$$

As a result, the epidemic naturally slows down once there are not enough susceptible (S) people left to catch the virus. When $R_e > 1$, each infectious person, on average, infects more than one other person, and the epidemic starts to grow. If $R_e < 1$, the disease usually fizzles out because each infected person does not create enough new cases to sustain the wave.

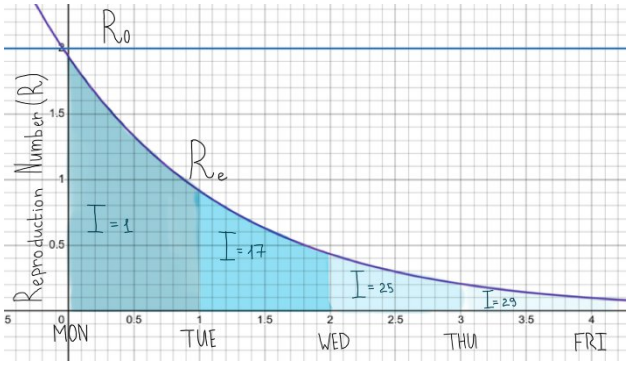

Figure 3: Exponential distribution and how the infectivity fell

In our class, R_e was changing $1.9 \rightarrow 0.9 \rightarrow 0.4 \rightarrow 0.2 \rightarrow 0.1$.

Effective number:

Mon: $R_e = 2 \times \frac{30}{31} \approx 1.9$

Tue: $R_e = 2 \times \frac{14}{31} \approx 0.9$

Wed: $R_e = 2 \times \frac{6}{31} \approx 0.4$

Thu: $R_e = 2 \times \frac{3}{31} \approx 0.2$

Fri: $R_e = 2 \times \frac{2}{31} \approx 0.1$

At first, most students were still susceptible, and the flu spread quickly, but as more of us became infected and recovered, there were fewer people left to catch the virus, and the outbreak weakened.

4. Epidemic distribution

But perhaps you have asked how the graph above was constructed? It can't just be a hyperbola with a random set of points?

In the SIR model, we assume that every healthy person gets sick at the rate of β and a sick person recovers at the rate of γ. The dynamics of S(t) are subject to the exponential distribution (Figure 3) - short infectious periods are more common, but longer ones are still possible. To work with predictions, that is, the speed of an event, there is $\lambda = \beta - \gamma$ average number of events per unit time

$$\text{Infection time T} \sim \text{Exp}(\lambda), \text{ where } \lambda = = \text{D day}^{-1}$$

$$\text{P(T>t)} = e^{(-\lambda t)}$$

For graph above I set these parameters:

$$R_e = R_0 \cdot \frac{S(t)}{N};\ S(t) = e^{(-0.4t)}$$

The exponential distribution is simple, but it can be replaced by the Erlang distribution to better capture how illness duration varies from person to person. Distribution density is

$$f(t) = e^{(-\lambda t)};\ F(t) = \int_0^t \frac{\lambda^k t^{k-1} e^{-\lambda t}}{(k-1)!} dx$$

k-numbers of stages (from Monday to Friday, 5)

λ-intensity ($\beta - \gamma$)

t-time (argument)

For Erlang distribution, function looks

$$f(t) = e^{(-0.4t)};\ F(t) = \int_0^t \frac{0.4^5 t^4 e^{-0.4t}}{(4)!}$$

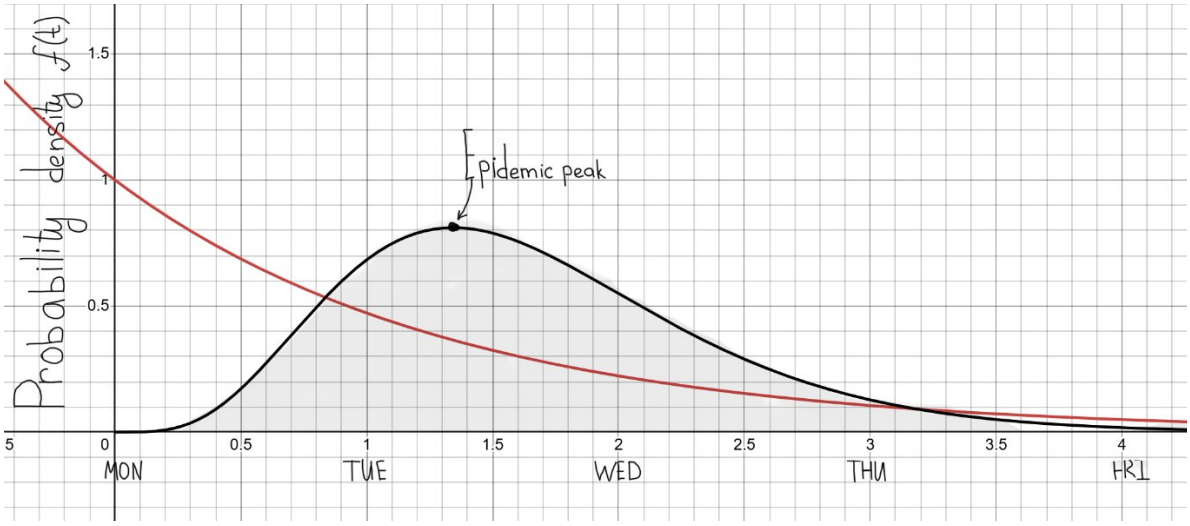

Figure 4: Erlang distribution

This means most students are sick for roughly the same number of days, while extremes—very brief or very long infectious periods—are much less common.

When there is no removal from the infectious group ($\gamma = 0$), the SIR model becomes the SI model, where individuals stay infected forever. This is similar to a chronic infection like HIV:

once someone is infected, they remain infectious for life—at least from the model's point of view.

5. How the flu spread in our school: the SIR model without birth and death

While our school battled the flu, no one died or enrolled anew. Epidemics like flu spread far faster than births or deaths, so we can safely ignore "vital dynamics." This simplified SIR model uses ordinary differential equations to track susceptibles (S), infected (I), and recovered (R).

$$\begin{cases} \frac{dS}{dN} = -\frac{\beta SI}{N} \\ \frac{dI}{dt} = \frac{\beta SI}{N} - \gamma I \\ \frac{dR}{dt} = \gamma I \end{cases}$$

These equations capture interconnected changes: healthy students get infected via contacts, infected recover over time, all tied to transmission rates and illness duration.

$$\begin{cases} \frac{dS}{dN} = \frac{0.8\times30\times1}{31} \\ \frac{dI}{dt} = \frac{0.8\times30\times1}{31} - 0.4\times1 \\ \frac{dR}{dt} = 0.4\times1 \end{cases} => \begin{cases} \frac{dS}{dN} \approx 0.8 \\ \frac{dI}{dt} \approx 0.4 \\ \frac{dR}{dt} = 0.4 \end{cases}$$

The number of those infected first grows rapidly when there are many healthy ones. But as the healthy are infected, fewer cases are being reported - and more is being added slowly. Eventually, the infected reach a peak and begin to fall until the outbreak subsides.

7. How it worked with the COVID 19

Early 2020: R0=2.5–3.0 drove exponential growth—China saw 80k cases by Feb. Global peak hit 10M+ cases by Jan 2021 as S (susceptible) dropped. Lockdowns slashed β by 70%, dropping effective R to <1. US hospitalizations peaked at 140k (Jan 2021) then crashed 90% by summer.

I(t) peaked then faded to near-zero in vaccinated regions—classic SIR behavior with ~770M total cases worldwide.

8. Why it matters to the world?

What started with a simple sneeze from the teacher in my classroom turned out to be a small-scale rehearsal of the huge epidemics described using the very same SIR model introduced by Kermack and McKendrick back in 1927. The point is how a single infected person can trigger a wave of disease, how this wave grows, reaches its peak, and then fades away, and how at the heart of all this lies one key idea — the number R_0, which decides the fate of any epidemic.

But behind my school-class story is a broader conclusion: the SIR model teaches us not just how to count the sick, but how interventions — from quarantine and online lessons to vaccination — break the chain of infection.