Lesson Objectives

1. Review (briefly) + continue extracting reaction rates from reaction equations
2. Connect reaction rate modeling to real-world example (disease spread)
3. Compare how population behaviors/interventions impact rate parameters
4. Hypothesize how model changes in non-ideal cases (real world inequities)

It is important as we consider how to give advice/make decisions/policy + consider engineering ethics.

Review

So far we have covered rate laws, which take the form of

\[ r_A = f(T, C_i, P, \text{etc}) \]

\[ \text{im important care about these bc they are impactful + tunable} \]
So we can write

\[ r_A = f(T) \cdot f(C) \]

\[ \text{①} \quad \text{②} \]

where

① Temperature dependence → rate constant
② Concentration dependence

③ derives from observations/lab (postulate from mechanism but only confirm via experiments)

④ use mass action (most often power law) to derive

⑤ can be elementary reactions where

① stoichiometric coefficients are identical to powers
② involves a single reaction step

So for example

\[ 2A \xrightleftharpoons{\kappa} B \]

\[ r_A = \frac{dC_A}{dt} = -2kC_A^2 \]
\[ \frac{dB}{dt} = dCa = kCa^2 \]

**SIR Models**

When modeling disease, SIR models are a common way to understand disease spread in a population. We have a system such that

\[ S \rightarrow I \rightarrow R \]

where

- \( S \) = susceptible
- \( I \) = infected
- \( R \) = removed (ideally by recovery)

and each is in units of fraction of the population.

\[ S + I + R = 1 \]
You can imagine the total population as a well-mixed batch reactor

where our initial fractions are

\[ S_i \approx \text{close to 1} \]
\[ I_i \approx \text{very small fraction} \]
\[ R_i \approx 0 \]

And you can imagine that the reactions governing disease spread can be described as

\[ S + I \xrightarrow{\beta} I + I \]

\( \beta \) can be thought of as a reactant akin to an auto-catalyst

where \( \beta \) – transmission rate constant

affected by

1. average frequency of contact between individuals (since space is not accounted for otherwise)
2. transmissibility of disease
2 \quad I \xrightarrow{\gamma} R

\text{can be thought of as catalyst deactivation}

where \( \gamma \) = recovery rate constant

1. \( \frac{1}{\gamma} \) = average duration of infection

So we can write reaction rates for each species w/ mass action kinetics:

1. **Reaction rate of S**

   \[
   r_S = \frac{dS}{dt} = -\beta SI
   \]

2. **Reaction rate of I**

   \[
   r_I = \frac{dI}{dt} = \beta SI - \gamma I
   \]

3. **Reaction rate of R**

   \[
   r_R = \frac{dR}{dt} = \gamma I
   \]
you can do a sanity check
we know that

\[ S + I + R = 1 \]

if you take the derivative

\[
\frac{d}{dt} [S + I + R] = \frac{d}{dt} (1)
\]

\[
\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0
\]

\[ (-\beta SI) + (\beta SI - \gamma I) + (\gamma I) = 0 \quad \checkmark \]

These fractions can be plotted over time--typically looks like

\[ \text{pop} \quad \text{time} \]

[Graph showing three curves S, I, and R over time with annotations]

this is the curve we talked about when we said "flatten the curve"
What assumptions does this model make? - no birth or death
- well mixed/no spatial heterogeneity/everybody interacts at the same rate
- everybody gets sick with the same probability & recovers at the same rate
- no reinfection
- no inequities

Analysis

We can think about how much a disease will spread based on some analysis of these equations

What causes infections to rise?

I > when \( \frac{dI}{dt} \)

\[
\frac{dI}{dt} = \beta S I - \gamma I
\]

\[= I [\beta S - \gamma] \]
So if we assume $I \neq 0$

$\frac{dI}{dt}$ if $\beta S - \gamma > 0$

$\beta S > \gamma$

$\frac{\beta S}{\gamma} > 1$

So infections will increase if

$\frac{\beta S}{\gamma} > 1$

This gives us the replacement number — expected # of people infected by a single infectious individual while they are infected

Each portion of the replacement number ($r$) has meaning:

$r = \frac{\beta S}{\gamma} \rightarrow$ produce $\beta S$ new infections per unit time

$\frac{1}{\gamma} \rightarrow$ be infectious for time $\frac{1}{\gamma}$
At time $t=0$, $S \approx 1$ and this gives us $R_0$ (R-naught)

\[ R_0 = \frac{\beta}{\gamma} \] commonly used in media

Example values:
- Seasonal flu = 1.3
- Ebola = 1.8
- COVID-19 = 4-10 (depending on variant)
- Chicken pox = 10 - 12
- Measles = 12 - 18

**ACTIVITY** → Think/Pair/Share, each 1/3rd of room gets one scenario

Let's think about this in the context of COVID

1. How do $\beta + \gamma$ change between the original vs delta/omicron variant?

   - $\text{B} + \text{omicron} > \text{B} + \text{delta} > \text{B} + \text{original}$ → omicron-delta spread more easily
   - $\text{Y} + \text{omicron} > \text{Y} + \text{delta} > \text{Y} + \text{original}$ → (most people) recover more quickly

2. How does having access to a vaccine change $\beta + \gamma$?

   - $\beta_{\text{vax}} < \beta_{\text{no vax}}$ → less spread
   - $\gamma_{\text{vax}} > \gamma_{\text{no vax}}$ → quicker recovery
How do social distancing + masking interventions affect $\beta + \gamma$?

$\beta_{\text{mask + SD}} < \beta_{\text{no mask or SD}} \quad \sim \quad \text{less spread}$

$\gamma$ is unaffected & $\Rightarrow \text{won't change recovery rate}$

Inequities in COVID-19

In worldwide pandemics like COVID-19 pandemic, systemic inequities can cause dramatic downstream impacts on disease spread + human health and survival.

What might some inequities be that could have this impact?
- access to healthcare/treatment/vaccines
- occupation
  - remote vs essential worker or in-person job
- environmental conditions
  - marginalized communities experience more negative affects from pollution + climate change, these can cause pre-existing conditions that make COVID more dangerous
- access to housing/type of housing
  - dictates exposure risk/frequency
- income
  - thus access to treatment + occupation $\Rightarrow$ wealth gap
- pre-existing conditions
  - can be related to external inequities (environmental injustice)
- discrimination

- not sharing resources (like vaccines) equally beyond the US
- different treatment by doctors for marginalized communities

During COVID-19, marginalized groups, such as racial + ethnic minorities faced increased risk of COVID-19 disease contraction, hospitalization, + death as a result of the compounded systemic inequities listed above (from CDC page). When developing disease interventions, preventions, + treatment (such as policy + analysis) it is important to consider those most vulnerable in a population.

The rate at which marginalized communities contracted or recovered from COVID-19 differs from that of those with more privilege. Thus, understanding disease spread in a community(s) might not be as simple as modeling disease spread in a population that assumes all individuals are equally at risk or interacting, susceptible, + recover at the same rate.

**Activity** - Brainstorm

How might the model/equations or model(s) change given the above?

- could have unique, interacting populations with different $\beta$+$\gamma$ values
- model w/more granular spatial distributions (global, country, state, country, city, communities)