McMaster University

Mathematics and Statistics

$$\int_M d\omega = \int_{\partial M} \omega$$

Mathematics 4MB3/6MB3
Mathematical Biology

Instructor: David Earn

Lecture 1
Epidemic Modelling Intro
Tuesday 3 September 2024

Course information

Course information

- Everyone should have received an e-mail.

Course information

- Everyone should have received an e-mail.
  - If not, please e-mail earn@math.mcmaster.ca now from the e-mail address that you use.

Course information

- Everyone should have received an e-mail.
  - If not, please e-mail earn@math.mcmaster.ca now from the e-mail address that you use.
- Course web page: `http://ms.mcmaster.ca/earn/4MB3`

Course information

- Everyone should have received an e-mail.
  - If not, please e-mail earn@math.mcmaster.ca now from the e-mail address that you use.
- Course web page: `http://ms.mcmaster.ca/earn/4MB3`
  - Click on "Course information".

Course information

- Everyone should have received an e-mail.
  - If not, please e-mail earn@math.mcmaster.ca now from the e-mail address that you use.
- Course web page: `http://ms.mcmaster.ca/earn/4MB3`
  - Click on "Course information".
  - Let's have a look now...

Mathematical Biology Research Seminar (MBRS)

Most weeks, there is a Mathematical Biology Research Seminar, which you are encouraged to attend if you are available.

- Where: **HH-410**
- When: **Thursdays, 2:30–3:20pm**
- Starts: **Thursday 12 September 2024**

If you would like to be on the e-mail distribution list for these seminars, please send me an e-mail (with MBRS in the subject line).

In-class polls: a form of participation

In-class polls: a form of participation

- Please log in (right now) to the childsmath web site: https://www.childsmath.ca/childsa/forms/main_login.php

In-class polls: a form of participation

- Please log in (right now) to the childsmath web site: https://www.childsmath.ca/childsa/forms/main_login.php
- Click on Math 4MB3.

In-class polls: a form of participation

- Please log in (right now) to the childsmath web site: https://www.childsmath.ca/childsa/forms/main_login.php
- Click on Math 4MB3.
- Click on Take Class Poll.

In-class polls: a form of participation

- Please log in (right now) to the childsmath web site: https://www.childsmath.ca/childsa/forms/main_login.php
- Click on Math 4MB3.
- Click on Take Class Poll.
- After selecting the person you think is your instructor, click the Submit button.

In-class polls: a form of participation

- Please log in (right now) to the childsmath web site: https://www.childsmath.ca/childsa/forms/main_login.php
- Click on Math 4MB3.
- Click on Take Class Poll.
- After selecting the person you think is your instructor, click the Submit button.
- Everybody done?

In-class polls: a form of participation

- Please log in (right now) to the childsmath web site: https://www.childsmath.ca/childsa/forms/main_login.php
- Click on Math 4MB3.
- Click on Take Class Poll.
- After selecting the person you think is your instructor, click the Submit button.
- Everybody done?
- Let's Deactivate the poll and View Results

Group formation

Most work in this course will be done in groups.

Group formation

Most work in this course will be done in groups.

- Form a group of 2 or 3 students **during the break TODAY.**

Group formation

Most work in this course will be done in groups.

- Form a group of 2 or 3 students **during the break TODAY.**
- **Exactly one** member of your group must `e-mail the instructor` **TODAY during the break:**

Group formation

Most work in this course will be done in groups.

- Form a group of 2 or 3 students **during the break TODAY.**
- **Exactly one** member of your group must `e-mail the instructor` **TODAY during the break:**
  - Include "Math 4MB3" and your proposed group name in the subject line.

Group formation

Most work in this course will be done in groups.

- Form a group of 2 or 3 students **during the break TODAY.**
- **Exactly one** member of your group must `e-mail the instructor` **TODAY during the break:**
  - Include "Math 4MB3" and your proposed group name in the subject line.
  - **Copy your message to all members of your proposed group so I have everyone's e-mail in the thread.**

Group formation

Most work in this course will be done in groups.

- Form a group of 2 or 3 students **during the break TODAY.**
- **Exactly one** member of your group must e-mail the instructor **TODAY during the break:**
  - Include "Math 4MB3" and your proposed group name in the subject line.
  - **Copy your message to all members of your proposed group so I have everyone's e-mail in the thread.**
- *Note: Instructor may change groups based on survey results.*

Online Surveys

You will be required to fill in online surveys during this course.

Online Surveys

You will be required to fill in online surveys during this course. Doing so in a timely manner contributes to your participation mark.

Online Surveys

You will be required to fill in online surveys during this course. Doing so in a timely manner contributes to your participation mark.

The first online survey has been posted:

Online Surveys

You will be required to fill in online surveys during this course. Doing so in a timely manner contributes to your participation mark.

The first online survey has been posted:

- Go to the Surveys page on the course web site.

Online Surveys

You will be required to fill in online surveys during this course. Doing so in a timely manner contributes to your participation mark.

The first online survey has been posted:

- Go to the Surveys page on the course web site.
- Follow the link for Background and Group formation Survey.
  - https://surveys.mcmaster.ca/limesurvey/index.php/746511

Online Surveys

You will be required to fill in online surveys during this course.
Doing so in a timely manner contributes to your participation mark.

The first online survey has been posted:

- Go to the Surveys page on the course web site.
- Follow the link for Background and Group formation Survey.
  - https://surveys.mcmaster.ca/limesurvey/index.php/746511
- Complete the survey ***TODAY***.

Online Surveys

You will be required to fill in online surveys during this course. Doing so in a timely manner contributes to your participation mark.

The first online survey has been posted:

- Go to the Surveys page on the course web site.
- Follow the link for Background and Group formation Survey.
  - `https://surveys.mcmaster.ca/limesurvey/index.php/746511`
- Complete the survey ***TODAY***.
- It should take only ~ 5 minutes.

Online Surveys

You will be required to fill in online surveys during this course. Doing so in a timely manner contributes to your participation mark.

The first online survey has been posted:

- Go to the Surveys page on the course web site.
- Follow the link for Background and Group formation Survey.
  - `https://surveys.mcmaster.ca/limesurvey/index.php/746511`
- Complete the survey ***TODAY***.
- It should take only ~ 5 minutes.
- Note that *surveys sometimes fail to save*.

Online Surveys

You will be required to fill in online surveys during this course. Doing so in a timely manner contributes to your participation mark.

The first online survey has been posted:

- Go to the Surveys page on the course web site.
- Follow the link for Background and Group formation Survey.
  - `https://surveys.mcmaster.ca/limesurvey/index.php/746511`
- Complete the survey ***TODAY***.
- It should take only ~ 5 minutes.
- Note that *surveys sometimes fail to save*.
  - Type long answers into a file first and paste them into the survey. Then you won't get as frustrated if it fails to save.

Software

Software

- **ASAP**, install the software discussed on the Software page on the course web site:

Software

- **ASAP**, install the software discussed on the Software page on the course web site:
  - $\LaTeX$

Software

- **ASAP**, install the software discussed on the Software page on the course web site:
  - $\LaTeX$ 
  - R

Software

- **ASAP**, install the software discussed on the Software page on the course web site:
  - LaTeX 
  - R
  - RStudio

Software

- **ASAP**, install the software discussed on the Software page on the course web site:
  - $\LaTeX$ 
  - R
  - RStudio
  - XPPAUT

Software

- **ASAP**, install the software discussed on the Software page on the course web site:
  - $\LaTeX$

  - R
  - RStudio
  - XPPAUT
  - Emacs

Software

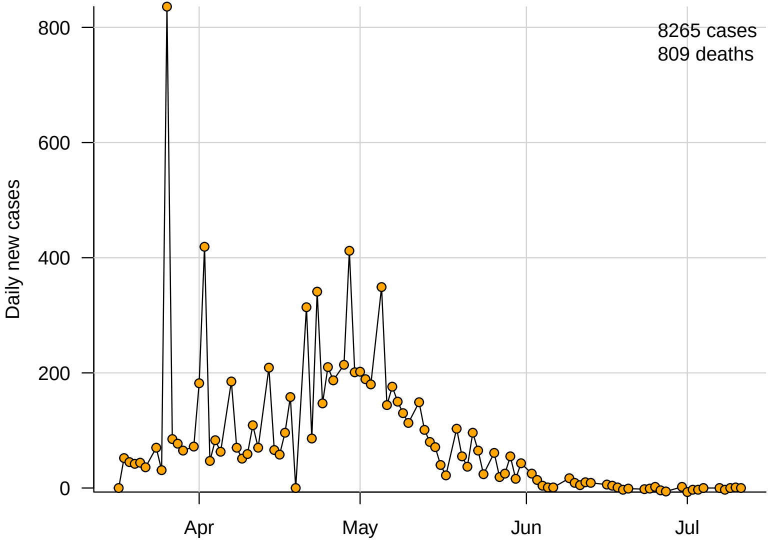
- **ASAP**, install the software discussed on the Software page on the course web site:
  - LaTeX

  - R
  - RStudio
  - XPPAUT
  - Emacs

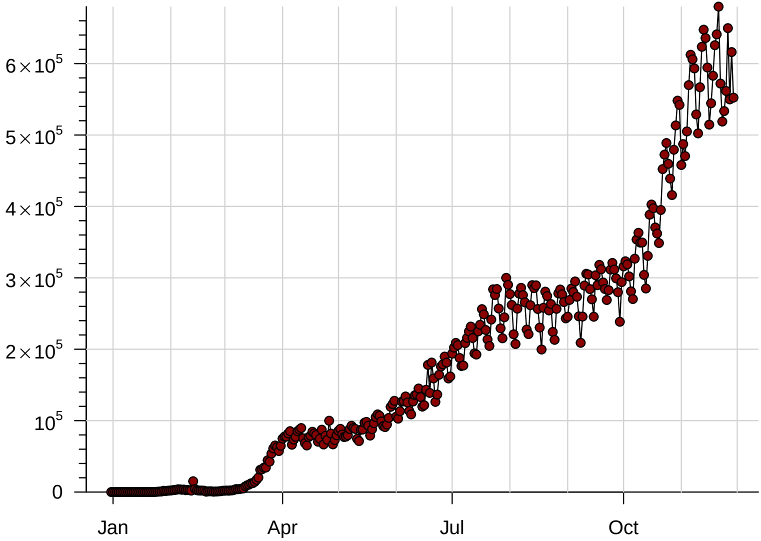
- *Note:* the Software page also contains some info about spell-checking and counting words in LaTeX documents.

Epidemic Modelling

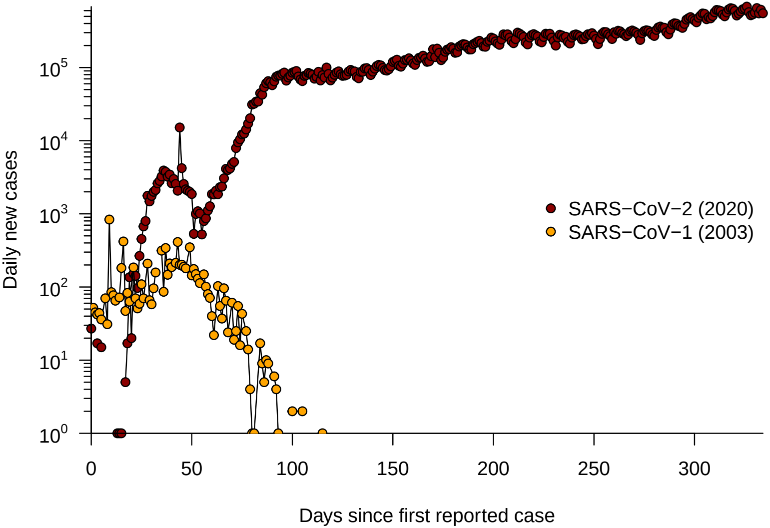
Daily SARS-CoV-1 in 2003 (Worldwide)



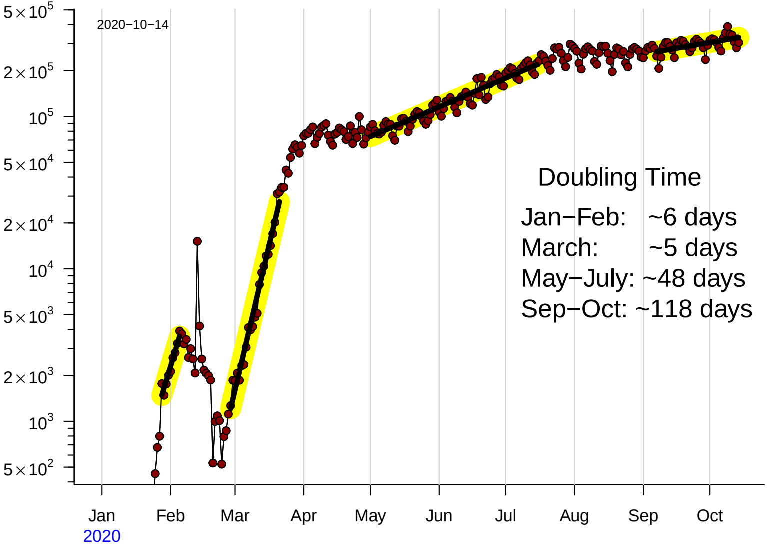
Daily SARS-CoV-2 in 2020 (Worldwide)



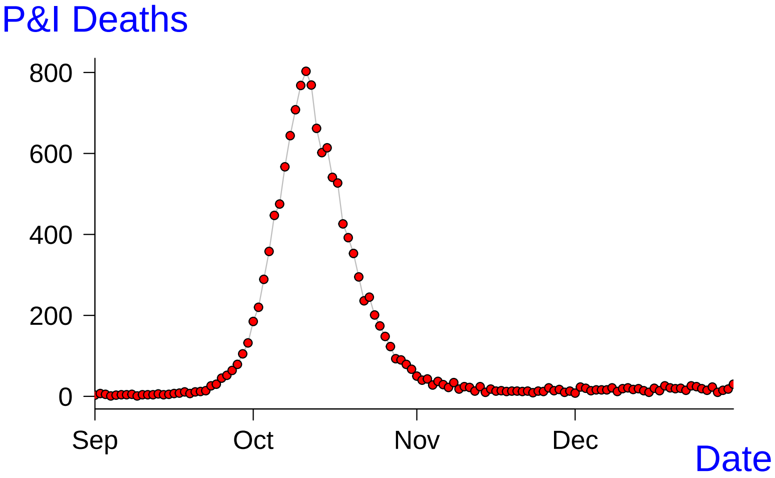
Daily SARS-CoV-1 vs SARS-CoV-2 (Worldwide)



Daily SARS-CoV-2 (Worldwide) exponential growth fits



Pneumonia & Influenza Mortality, Philadelphia, 1918



Modelling challenge

Develop a model that helps us understand the graph on the previous slide, based on mechanisms of disease spread.

Modelling challenge

Develop a model that helps us understand the graph on the previous slide, based on mechanisms of disease spread.

- Only one variable is observed (P&I deaths per day) so construct a model containing only one variable.

Modelling challenge

Develop a model that helps us understand the graph on the previous slide, based on mechanisms of disease spread.

- Only one variable is observed (P&I deaths per day) so construct a model containing only one variable.
- Think about how disease spreads and express your thoughts with mathematical notation.

Modelling challenge

Develop a model that helps us understand the graph on the previous slide, based on mechanisms of disease spread.

- Only one variable is observed (P&I deaths per day) so construct a model containing only one variable.
- Think about how disease spreads and express your thoughts with mathematical notation.
- Derive a differential equation that models the process of disease transmission.

Modelling challenge

Develop a model that helps us understand the graph on the previous slide, based on mechanisms of disease spread.

- Only one variable is observed (P&I deaths per day) so construct a model containing only one variable.
- Think about how disease spreads and express your thoughts with mathematical notation.
- Derive a differential equation that models the process of disease transmission.
- Analyze the model and determine its strengths and weaknesses/limitations.

Make (Biological) Assumptions Clear

Make (Biological) Assumptions Clear

1. *Assume* the disease is transmitted by

Make (Biological) Assumptions Clear

1. *Assume* the disease is transmitted by contact between an infected individual and a susceptible individual.

Make (Biological) Assumptions Clear

1. *Assume* the disease is transmitted by contact between an infected individual and a susceptible individual.
2. *Assume* the latent period (delay between being infected and becoming infectious) is

Make (Biological) Assumptions Clear

1. *Assume* the disease is transmitted by contact between an infected individual and a susceptible individual.
2. *Assume* the latent period (delay between being infected and becoming infectious) is so short that it can be ignored (technically assume it is zero).

Make (Biological) Assumptions Clear

1. *Assume* the disease is transmitted by contact between an infected individual and a susceptible individual.
2. *Assume* the latent period (delay between being infected and becoming infectious) is so short that it can be ignored (technically assume it is zero).
3. *Assume* all members of the population are identical and respond identically to the disease.

Make (Biological) Assumptions Clear

1. *Assume* the disease is transmitted by contact between an infected individual and a susceptible individual.
2. *Assume* the latent period (delay between being infected and becoming infectious) is so short that it can be ignored (technically assume it is zero).
3. *Assume* all members of the population are identical and respond identically to the disease. In particular, all susceptible individuals are equally susceptible and all infected individuals are equally infectious.

Make (Biological) Assumptions Clear

1. *Assume* the disease is transmitted by contact between an infected individual and a susceptible individual.
2. *Assume* the latent period (delay between being infected and becoming infectious) is so short that it can be ignored (technically assume it is zero).
3. *Assume* all members of the population are identical and respond identically to the disease. In particular, all susceptible individuals are equally susceptible and all infected individuals are equally infectious.
4. *Assume* the population size is fixed during the epidemic

Make (Biological) Assumptions Clear

1. *Assume* the disease is transmitted by contact between an infected individual and a susceptible individual.
2. *Assume* the latent period (delay between being infected and becoming infectious) is so short that it can be ignored (technically assume it is zero).
3. *Assume* all members of the population are identical and respond identically to the disease. In particular, all susceptible individuals are equally susceptible and all infected individuals are equally infectious.
4. *Assume* the population size is fixed during the epidemic, *i.e.*, ignore births, migration, and deaths from causes other than the disease

Make (Biological) Assumptions Clear

1. *Assume* the disease is transmitted by contact between an infected individual and a susceptible individual.
2. *Assume* the latent period (delay between being infected and becoming infectious) is so short that it can be ignored (technically assume it is zero).
3. *Assume* all members of the population are identical and respond identically to the disease. In particular, all susceptible individuals are equally susceptible and all infected individuals are equally infectious.
4. *Assume* the population size is fixed during the epidemic, *i.e.*, ignore births, migration, and deaths from causes other than the disease, and count individuals who have died from the disease as part of the population.

About Assumptions. . .

About Assumptions. . .

- Note that the first assumption on the previous slide is actually correct.

About Assumptions. . .

- Note that the first assumption on the previous slide is actually correct.
- The other assumptions are wrong,

About Assumptions. . .

- Note that the first assumption on the previous slide is actually correct.
- The other assumptions are wrong, but are reasonable approximations.

About Assumptions. . .

- Note that the first assumption on the previous slide is actually correct.
- The other assumptions are wrong, but are reasonable approximations.
- It is best to *start as simple as possible and add complexity later*, in order to:

About Assumptions. . .

- Note that the first assumption on the previous slide is actually correct.
- The other assumptions are wrong, but are reasonable approximations.
- It is best to *start as simple as possible and add complexity later*, in order to:
    - obtain a model that actually succeeds in explaining the data with as few ingredients as possible;

About Assumptions...

- Note that the first assumption on the previous slide is actually correct.
- The other assumptions are wrong, but are reasonable approximations.
- It is best to *start as simple as possible and add complexity later,* in order to:
  - obtain a model that actually succeeds in explaining the data with as few ingredients as possible;
  - check that inferences we draw from our model(s) are robust to the inclusion of more biological details/realism.

What variables should we include in our model?

What variables should we include in our model?

- Independent variable:

What variables should we include in our model?

- Independent variable: time (t)

What variables should we include in our model?

- Independent variable: time (t)
- Dependent variable:

What variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: Many options, *e.g.*,

What variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: Many options, *e.g.*,
  - Incidence (number of new infections per unit time)

What variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: Many options, *e.g.*,
  - Incidence (number of new infections per unit time)
  - Prevalence (total current number of infected individuals)

What variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: Many options, *e.g.*,
  - Incidence (number of new infections per unit time)
  - Prevalence (total current number of infected individuals)
  - Death rate (number of deaths per unit time)

What variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: Many options, *e.g.*,
  - Incidence (number of new infections per unit time)
  - Prevalence (total current number of infected individuals)
  - Death rate (number of deaths per unit time)
  - Death toll (number of deaths so far)

What variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: Many options, *e.g.*,
  - Incidence (number of new infections per unit time)
  - Prevalence (total current number of infected individuals)
  - Death rate (number of deaths per unit time)
  - Death toll (number of deaths so far)
- So, what would be best?

What variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: Many options, *e.g.*,
  - Incidence (number of new infections per unit time)
  - Prevalence (total current number of infected individuals)
  - Death rate (number of deaths per unit time)
  - Death toll (number of deaths so far)
- So, what would be best?
- Not deaths, because

What variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: Many options, *e.g.*,
  - Incidence (number of new infections per unit time)
  - Prevalence (total current number of infected individuals)
  - Death rate (number of deaths per unit time)
  - Death toll (number of deaths so far)
- So, what would be best?
- Not deaths, because whether or not you die may be unrelated to how much you transmit.

What variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: Many options, *e.g.*,
  - Incidence (number of new infections per unit time)
  - Prevalence (total current number of infected individuals)
  - Death rate (number of deaths per unit time)
  - Death toll (number of deaths so far)
- So, what would be best?
- Not deaths, because whether or not you die may be unrelated to how much you transmit.
- But deaths are what we observe!

What variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: Many options, *e.g.*,
  - Incidence (number of new infections per unit time)
  - Prevalence (total current number of infected individuals)
  - Death rate (number of deaths per unit time)
  - Death toll (number of deaths so far)
- So, what would be best?
- Not deaths, because whether or not you die may be unrelated to how much you transmit.
- But deaths are what we observe! What to do?!?

What variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: Many options, *e.g.*,
  - Incidence (number of new infections per unit time)
  - Prevalence (total current number of infected individuals)
  - Death rate (number of deaths per unit time)
  - Death toll (number of deaths so far)
- So, what would be best?
- Not deaths, because whether or not you die may be unrelated to how much you transmit.
- But deaths are what we observe! What to do?!?
- Make another assumption...

Additional assumption(s)

Additional assumption(s)

- We actually want to know incidence or prevalence, but we observe deaths.

Additional assumption(s)

- We actually want to know incidence or prevalence, but we observe deaths.
- Under what circumstances would daily deaths be a good estimate of incidence?

Additional assumption(s)

- We actually want to know incidence or prevalence, but we observe deaths.
- Under what circumstances would daily deaths be a good estimate of incidence? (*i.e.*, What must we assume in addition to the assumptions we have already made.)

Additional assumption(s)

- We actually want to know incidence or prevalence, but we observe deaths.
- Under what circumstances would daily deaths be a good estimate of incidence? (*i.e.*, What must we assume in addition to the assumptions we have already made.)
    5. *Assume* that the time from infection to death is

Additional assumption(s)

- We actually want to know incidence or prevalence, but we observe deaths.
- Under what circumstances would daily deaths be a good estimate of incidence? (*i.e.*, What must we assume in addition to the assumptions we have already made.)
    5. *Assume* that the time from infection to death is exactly the same (a certain number of days) for every individual who dies.

Additional assumption(s)

- We actually want to know incidence or prevalence, but we observe deaths.
- Under what circumstances would daily deaths be a good estimate of incidence? (*i.e.*, What must we assume in addition to the assumptions we have already made.)
  5. *Assume* that the time from infection to death is exactly the same (a certain number of days) for every individual who dies.
  6. *Assume* that the probability of dying from the disease is

Additional assumption(s)

- We actually want to know incidence or prevalence, but we observe deaths.
- Under what circumstances would daily deaths be a good estimate of incidence? (*i.e.*, What must we assume in addition to the assumptions we have already made.)
  5. *Assume* that the time from infection to death is exactly the same (a certain number of days) for every individual who dies.
  6. *Assume* that the probability of dying from the disease is the same for every individual who is infected.

Additional assumption(s)

- We actually want to know incidence or prevalence, but we observe deaths.
- Under what circumstances would daily deaths be a good estimate of incidence? (*i.e.*, What must we assume in addition to the assumptions we have already made.)
  5. *Assume* that the time from infection to death is exactly the same (a certain number of days) for every individual who dies.
  6. *Assume* that the probability of dying from the disease is the same for every individual who is infected.
- Then daily death counts are

Additional assumption(s)

- We actually want to know incidence or prevalence, but we observe deaths.
- Under what circumstances would daily deaths be a good estimate of incidence? (*i.e.*, What must we assume in addition to the assumptions we have already made.)
  5. *Assume* that the time from infection to death is exactly the same (a certain number of days) for every individual who dies.
  6. *Assume* that the probability of dying from the disease is the same for every individual who is infected.
- Then daily death counts are proportional to daily incidence a certain number of days in the past,

Additional assumption(s)

- We actually want to know incidence or prevalence, but we observe deaths.
- Under what circumstances would daily deaths be a good estimate of incidence? (*i.e.*, What must we assume in addition to the assumptions we have already made.)
  5. *Assume* that the time from infection to death is exactly the same (a certain number of days) for every individual who dies.
  6. *Assume* that the probability of dying from the disease is the same for every individual who is infected.
- Then daily death counts are proportional to daily incidence a certain number of days in the past, *i.e.*, the "mortality curve" that we observe is a translated and scaled version of the "epidemic curve" (new cases per day).

So. . . what variables should we include in our model?

So. . . what variables should we include in our model?

- Independent variable:

So. . . what variables should we include in our model?

- Independent variable: time (t)

So. . . what variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: one of:

So. . . what variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: one of:
  - Incidence (number of new infections per unit time)
  - Prevalence (total current number of infected individuals)

So. . . what variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: one of:
  - Incidence (number of new infections per unit time)
  - Prevalence (total current number of infected individuals)
- Which one?

So. . . what variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: one of:
  - Incidence (number of new infections per unit time)
  - Prevalence (total current number of infected individuals)
- Which one?
- Choose prevalence (I) because

So. . . what variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: one of:
  - Incidence (number of new infections per unit time)
  - Prevalence (total current number of infected individuals)
- Which one?
- Choose prevalence (I) because anybody who is currently infectious can infect others,

So. . . what variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: one of:
  - Incidence (number of new infections per unit time)
  - Prevalence (total current number of infected individuals)
- Which one?
- Choose prevalence (I) because anybody who is currently infectious can infect others, so it will probably be easier to formulate a transmission model based on prevalence.

So. . . what variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: one of:
  - Incidence (number of new infections per unit time)
  - Prevalence (total current number of infected individuals)
- Which one?
- Choose prevalence (I) because anybody who is currently infectious can infect others, so it will probably be easier to formulate a transmission model based on prevalence. (Try not to lose sight of underlying biological mechanisms.)

So. . . what variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: one of:
  - Incidence (number of new infections per unit time)
  - Prevalence (total current number of infected individuals)
- Which one?
- Choose prevalence (I) because anybody who is currently infectious can infect others, so it will probably be easier to formulate a transmission model based on prevalence.
  (Try not to lose sight of underlying biological mechanisms.)
- But our mortality curve is related to incidence, not prevalence!?!

So. . . what variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: one of:
  - Incidence (number of new infections per unit time)
  - Prevalence (total current number of infected individuals)
- Which one?
- Choose prevalence (I) because anybody who is currently infectious can infect others, so it will probably be easier to formulate a transmission model based on prevalence.
  (Try not to lose sight of underlying biological mechanisms.)
- But our mortality curve is related to incidence, not prevalence!?! Argh. What to do?!?

So. . . what variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: one of:
  - Incidence (number of new infections per unit time)
  - Prevalence (total current number of infected individuals)
- Which one?
- Choose prevalence (I) because anybody who is currently infectious can infect others, so it will probably be easier to formulate a transmission model based on prevalence.
  (Try not to lose sight of underlying biological mechanisms.)
- But our mortality curve is related to incidence, not prevalence!?! Argh. What to do?!?
- Let's work with prevalence and see how it works out.

So. . . what variables should we include in our model?

- Independent variable: time (t)
- Dependent variable: one of:
  - Incidence (number of new infections per unit time)
  - Prevalence (total current number of infected individuals)
- Which one?
- Choose prevalence (I) because anybody who is currently infectious can infect others, so it will probably be easier to formulate a transmission model based on prevalence. (Try not to lose sight of underlying biological mechanisms.)
- But our mortality curve is related to incidence, not prevalence!?! Argh. What to do?!?
- Let's work with prevalence and see how it works out. Maybe we'll be able to derive the incidence curve from a model based on prevalence.

Notational note

Notational note

- We use I for prevalence because prevalence is the number of infected individuals.

Notational note

- We use I for prevalence because prevalence is the number of infected individuals.

- So, let's try to write down a model...

A first (naïve) attempt at an epidemic model

A first (naïve) attempt at an epidemic model

- Variables: time t, prevalence $I(t)$

A first (naïve) attempt at an epidemic model

- Variables: time t, prevalence $I(t)$
- How does I increase?

A first (naïve) attempt at an epidemic model

- Variables: time t, prevalence $I(t)$
- How does I increase?
- Start with I_0 infected individuals at time $t = 0$. What happens for $t > 0$.

A first (naïve) attempt at an epidemic model

- Variables: time t, prevalence $I(t)$
- How does I increase?
- Start with I_0 infected individuals at time $t = 0$. What happens for $t > 0$.
- Let $B =$ average number of contacts with susceptible individuals that lead to a new infective per unit time per infective in the population (and suppose B is constant). Then

A first (naïve) attempt at an epidemic model

- Variables: time t, prevalence $I(t)$
- How does I increase?
- Start with I_0 infected individuals at time $t = 0$. What happens for $t > 0$.
- Let $B =$ average number of contacts with susceptible individuals that lead to a new infective per unit time per infective in the population (and suppose B is constant). Then

$$I(t + \Delta t) \simeq$$

A first (naïve) attempt at an epidemic model

- Variables: time t, prevalence $I(t)$
- How does I increase?
- Start with I_0 infected individuals at time $t = 0$. What happens for $t > 0$.
- Let $B =$ average number of contacts with susceptible individuals that lead to a new infective per unit time per infective in the population (and suppose B is constant). Then

$$I(t + \Delta t) \simeq I(t) +$$

A first (naïve) attempt at an epidemic model

- Variables: time t, prevalence $I(t)$
- How does I increase?
- Start with I_0 infected individuals at time $t = 0$. What happens for $t > 0$.
- Let $B =$ average number of contacts with susceptible individuals that lead to a new infective per unit time per infective in the population (and suppose B is constant). Then

$$I(t + \Delta t) \simeq I(t) + B\, I(t)\Delta t$$

A first (naïve) attempt at an epidemic model

- Variables: time t, prevalence $I(t)$
- How does I increase?
- Start with I_0 infected individuals at time $t = 0$. What happens for $t > 0$.
- Let B = average number of contacts with susceptible individuals that lead to a new infective per unit time per infective in the population (and suppose B is constant). Then

$$I(t + \Delta t) \simeq I(t) + B\, I(t)\Delta t$$

- In the limit $\Delta t \to 0$, we have

A first (naïve) attempt at an epidemic model

- Variables: time t, prevalence $I(t)$
- How does I increase?
- Start with I_0 infected individuals at time $t = 0$. What happens for $t > 0$.
- Let $B =$ average number of contacts with susceptible individuals that lead to a new infective per unit time per infective in the population (and suppose B is constant). Then

$$I(t + \Delta t) \simeq I(t) + B\, I(t)\Delta t$$

- In the limit $\Delta t \to 0$, we have

$$\frac{dI}{dt} =$$

A first (naïve) attempt at an epidemic model

- Variables: time t, prevalence $I(t)$
- How does I increase?
- Start with I_0 infected individuals at time $t = 0$. What happens for $t > 0$.
- Let $B =$ average number of contacts with susceptible individuals that lead to a new infective per unit time per infective in the population (and suppose B is constant). Then

$$I(t + \Delta t) \simeq I(t) + B\, I(t)\Delta t$$

- In the limit $\Delta t \to 0$, we have

$$\frac{dI}{dt} = BI$$

A first (naïve) attempt at an epidemic model

- Variables: time t, prevalence $I(t)$
- How does I increase?
- Start with I_0 infected individuals at time $t = 0$. What happens for $t > 0$.
- Let $B =$ average number of contacts with susceptible individuals that lead to a new infective per unit time per infective in the population (and suppose B is constant). Then

$$I(t + \Delta t) \simeq I(t) + B\, I(t)\Delta t$$

- In the limit $\Delta t \to 0$, we have

$$\frac{dI}{dt} = BI \quad \Longrightarrow$$

A first (naïve) attempt at an epidemic model

- Variables: time t, prevalence $I(t)$
- How does I increase?
- Start with I_0 infected individuals at time $t = 0$. What happens for $t > 0$.
- Let $B =$ average number of contacts with susceptible individuals that lead to a new infective per unit time per infective in the population (and suppose B is constant). Then

$$I(t + \Delta t) \simeq I(t) + B\,I(t)\Delta t$$

- In the limit $\Delta t \to 0$, we have

$$\frac{dI}{dt} = BI \quad \Longrightarrow \quad I(t) = I_0 e^{Bt}$$

Beware: implicit assumptions that should be explicit

Beware: implicit assumptions that should be explicit

- Ignored discrete nature of individuals when taking limit.

Beware: implicit assumptions that should be explicit

- Ignored discrete nature of individuals when taking limit.
- *Ignored finite infectious periods!*

Beware: implicit assumptions that should be explicit

- Ignored discrete nature of individuals when taking limit.
- *Ignored finite infectious periods!*
    - Sometimes it isn't obvious that we've made some assumptions until after we see what the model predicts.

How can we tell if our model is good?

How can we tell if our model is good?

- Compare model predictions with data.

How can we tell if our model is good?

- Compare model predictions with data.
- What is the best way to do that?

How can we tell if our model is good?

- Compare model predictions with data.
- What is the best way to do that?
- Depends on what predictions we're trying to test.

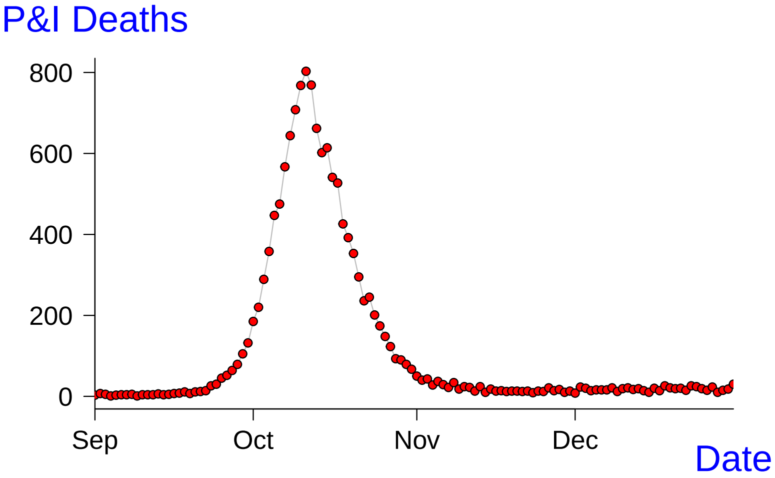
How can we tell if our model is good?

- Compare model predictions with data.
- What is the best way to do that?
- Depends on what predictions we're trying to test.
- Model predicts exponential growth.
  How should we test that prediction?

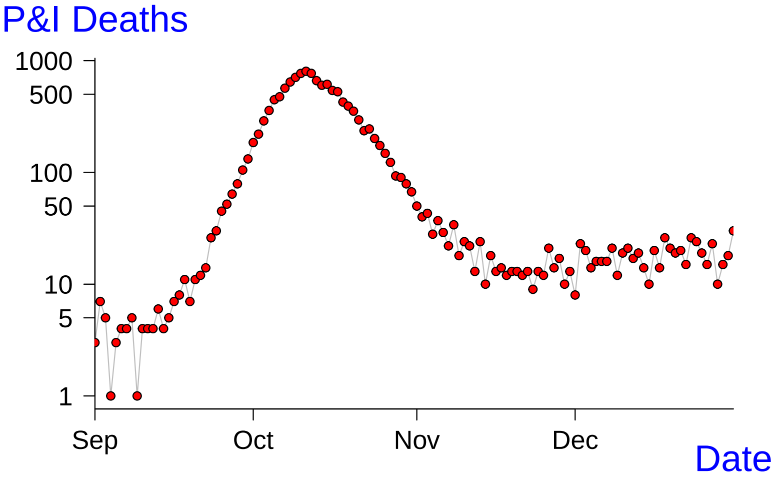
How can we tell if our model is good?

- Compare model predictions with data.
- What is the best way to do that?
- Depends on what predictions we're trying to test.
- Model predicts exponential growth.
  How should we test that prediction?
- Transforming the data might help.

Original data: P&I Mortality, Philadelphia, 1918



Logarithmic scale: P&I Mortality, Philadelphia, 1918



Parameter estimation

How can we estimate the model parameters, I_0 and B, from the P&I data?

Parameter estimation

How can we estimate the model parameters, I_0 and B, from the P&I data?

- Fit a straight line through the part of the logarithmic mortality curve that looks straight.

Parameter estimation

How can we estimate the model parameters, I_0 and B, from the P&I data?

- Fit a straight line through the part of the logarithmic mortality curve that looks straight.
- The slope of the line is B.

Parameter estimation

How can we estimate the model parameters, I_0 and B, from the P&I data?

- Fit a straight line through the part of the logarithmic mortality curve that looks straight.
- The slope of the line is B.
- The "intercept" is $\log I_0$.

Parameter estimation

How can we estimate the model parameters, I_0 and B, from the P&I data?

- Fit a straight line through the part of the logarithmic mortality curve that looks straight.
- The slope of the line is B.
- The "intercept" is $\log I_0$.
  - "Intercept" in quotes because we need to define $t = 0$ as the time when exponential growth begins.

Parameter estimation

How can we estimate the model parameters, I_0 and B, from the P&I data?

- Fit a straight line through the part of the logarithmic mortality curve that looks straight.
- The slope of the line is B.
- The "intercept" is $\log I_0$.
  - "Intercept" in quotes because we need to define $t = 0$ as the time when exponential growth begins.
- *Note:* Parameter estimation is, in general, a very tricky business and deserves a great deal of attention.

Parameter estimation

How can we estimate the model parameters, I_0 and B, from the P&I data?

- Fit a straight line through the part of the logarithmic mortality curve that looks straight.
- The slope of the line is B.
- The "intercept" is $\log I_0$.
  - "Intercept" in quotes because we need to define $t = 0$ as the time when exponential growth begins.
- *Note:* Parameter estimation is, in general, a very tricky business and deserves a great deal of attention.

Earn, Park, Bolker (2024) "Fitting Epidemic Models to Data..." *Bull. Math. Biol.* **86**, 109

Naïve epidemic model

Naïve epidemic model

- Variables: time t, prevalence $I(t)$

Naïve epidemic model

- Variables: time t, prevalence $I(t)$
- Parameter $B =$ average number of contacts with susceptible individuals that lead to a new infective per unit time per infective in the population

Naïve epidemic model

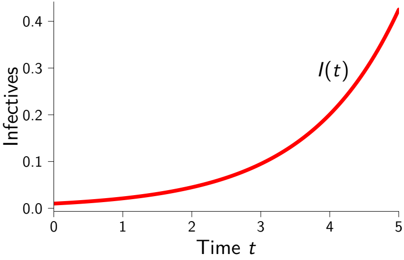
- Variables: time t, prevalence $I(t)$
- Parameter $B =$ average number of contacts with susceptible individuals that lead to a new infective per unit time per infective in the population

$$\frac{dI}{dt} = BI \quad \Longrightarrow \quad I(t) = I_0 e^{Bt}$$

Naïve epidemic model

- Variables: time t, prevalence $I(t)$
- Parameter $B =$ average number of contacts with susceptible individuals that lead to a new infective per unit time per infective in the population

$$\frac{dI}{dt} = BI \quad \Longrightarrow \quad I(t) = I_0 e^{Bt}$$



Naïve model: the good and the bad

Naïve model: the good and the bad

- Good:

Naïve model: the good and the bad

- Good:
  - Makes clear predictions

Naïve model: the good and the bad

- Good:
  - Makes clear predictions
  - Predictions can be tested

Naïve model: the good and the bad

- Good:
  - Makes clear predictions
  - Predictions can be tested
  - Estimation of parameter (B) is easy

Naïve model: the good and the bad

- Good:
  - Makes clear predictions
  - Predictions can be tested
  - Estimation of parameter (B) is easy
    - B is the slope of the straight portion of the epidemic curve on the log scale.

Naïve model: the good and the bad

- Good:
  - Makes clear predictions
  - Predictions can be tested
  - Estimation of parameter (B) is easy
    - B is the slope of the straight portion of the epidemic curve on the log scale. (Why?)

Naïve model: the good and the bad

- Good:
  - Makes clear predictions
  - Predictions can be tested
  - Estimation of parameter (B) is easy
    - B is the slope of the straight portion of the epidemic curve on the log scale. (Why?)
    - Remember we are imagining that the mortality curve is equivalent to the epidemic curve after translation and scaling.

Naïve model: the good and the bad

- Good:
  - Makes clear predictions
  - Predictions can be tested
  - Estimation of parameter (B) is easy
    - B is the slope of the straight portion of the epidemic curve on the log scale. (Why?)
    - Remember we are imagining that the mortality curve is equivalent to the epidemic curve after translation and scaling.
    - Why do translation and scaling not affect the estimate of B?

Naïve model: the good and the bad

- Good:
  - Makes clear predictions
  - Predictions can be tested
  - Estimation of parameter (B) is easy
    - B is the slope of the straight portion of the epidemic curve on the log scale. (Why?)
    - Remember we are imagining that the mortality curve is equivalent to the epidemic curve after translation and scaling.
    - Why do translation and scaling not affect the estimate of B? Assignment 2...

Naïve model: the good and the bad

- Good:
  - Makes clear predictions
  - Predictions can be tested
  - Estimation of parameter (B) is easy
    - B is the slope of the straight portion of the epidemic curve on the log scale. (Why?)
    - Remember we are imagining that the mortality curve is equivalent to the epidemic curve after translation and scaling.
    - Why do translation and scaling not affect the estimate of B? Assignment 2...
- Bad:

Naïve model: the good and the bad

- Good:
  - Makes clear predictions
  - Predictions can be tested
  - Estimation of parameter (B) is easy
    - B is the slope of the straight portion of the epidemic curve on the log scale. (Why?)
    - Remember we are imagining that the mortality curve is equivalent to the epidemic curve after translation and scaling.
    - Why do translation and scaling not affect the estimate of B? Assignment 2...
- Bad:
  - Model is consistent only with exponential growth phase.

Naïve model: the good and the bad

- Good:
  - Makes clear predictions
  - Predictions can be tested
  - Estimation of parameter (B) is easy
    - B is the slope of the straight portion of the epidemic curve on the log scale. (Why?)
    - Remember we are imagining that the mortality curve is equivalent to the epidemic curve after translation and scaling.
    - Why do translation and scaling not affect the estimate of B? Assignment 2...
- Bad:
  - Model is consistent only with exponential growth phase.
  - Absurd long-term prediction: unbounded growth in $I(t)$

Naïve model: the good and the bad

- Good:
  - Makes clear predictions
  - Predictions can be tested
  - Estimation of parameter (B) is easy
    - B is the slope of the straight portion of the epidemic curve on the log scale. (Why?)
    - Remember we are imagining that the mortality curve is equivalent to the epidemic curve after translation and scaling.
    - Why do translation and scaling not affect the estimate of B? Assignment 2...
- Bad:
  - Model is consistent only with exponential growth phase.
  - Absurd long-term prediction: unbounded growth in $I(t)$
    - Implicitly assumed that population size $N = \infty$.

How can we improve our model?

How can we improve our model?

- Insist that population size is finite ($N < \infty$).

How can we improve our model?

- Insist that population size is finite ($N < \infty$).
- Keep track of both infectives $I(t)$ and susceptibles $S(t)$.

How can we improve our model?

- Insist that population size is finite ($N < \infty$).
- Keep track of both infectives $I(t)$ and susceptibles $S(t)$.
- Assume individuals who are *not infected* are susceptible:

$$I(t) + S(t) = N = \text{constant}.$$

New model parameter(s)?

New model parameter(s)?

- $B =$ average number of contacts with susceptible individuals that lead to a new infective *per* unit time *per* infective

New model parameter(s)?

- $B =$ average number of contacts with susceptible individuals that lead to a new infective *per* unit time *per* infective
- In the naïve model, we assumed $B =$ constant.

New model parameter(s)?

- $B =$ average number of contacts with susceptible individuals that lead to a new infective *per* unit time *per* infective
- In the naïve model, we assumed $B =$ constant.
  Is B really constant?

New model parameter(s)?

- $B =$ average number of contacts with susceptible individuals that lead to a new infective *per* unit time *per* infective
- In the naïve model, we assumed $B =$ constant.
  Is B really constant?
- B depends on how many susceptibles there are.

New model parameter(s)?

- $B =$ average number of contacts with susceptible individuals that lead to a new infective *per* unit time *per* infective
- In the naïve model, we assumed $B =$ constant.
  Is B really constant?
- B depends on how many susceptibles there are.
- $B =$

New model parameter(s)?

- $B =$ average number of contacts with susceptible individuals that lead to a new infective *per* unit time *per* infective
- In the naïve model, we assumed $B =$ constant. Is B really constant?
- B depends on how many susceptibles there are.
- $B = \beta S(t)$

New model parameter(s)?

- $B =$ average number of contacts with susceptible individuals that lead to a new infective *per* unit time *per* infective
- In the naïve model, we assumed $B =$ constant.
  Is B really constant?
- B depends on how many susceptibles there are.
- $B = \beta S(t)$
- $\beta =$

New model parameter(s)?

- $B =$ average number of contacts with susceptible individuals that lead to a new infective *per* unit time *per* infective
- In the naïve model, we assumed $B =$ constant.
  Is B really constant?
- B depends on how many susceptibles there are.
- $B = \beta S(t)$
- $\beta =$ average number of contacts between susceptibles and infectives that lead to a new infective
  per unit time
  per infective
  per susceptible

New model parameter(s)?

- $B =$ average number of contacts with susceptible individuals that lead to a new infective *per* unit time *per* infective
- In the naïve model, we assumed $B =$ constant.
  Is B really constant?
- B depends on how many susceptibles there are.
- $B = \beta S(t)$
- $\beta =$ average number of contacts between susceptibles and infectives that lead to a new infective
  - *per* unit time
  - *per* infective
  - <u>*per* susceptible</u>
- β is called the ***transmission rate***.

Revised epidemic model: "SI model"

$$\frac{dI}{dt} = \beta S(t)I(t)$$

Revised epidemic model: “SI model”

$$\frac{dI}{dt} = \beta S(t)I(t)$$

- Two state variables. One equation.

Revised epidemic model: "SI model"

$$\frac{dI}{dt} = \beta S(t)I(t)$$

- Two state variables. One equation. Problem?

Revised epidemic model: “SI model”

$$\frac{dI}{dt} = \beta S(t)I(t)$$

- Two state variables. One equation. Problem? No:

Revised epidemic model: "SI model"

$$\frac{dI}{dt} = \beta S(t)I(t)$$

- Two state variables. One equation. Problem? No:

$$\frac{dS}{dt} = -\beta S(t)I(t)$$

Revised epidemic model: "SI model"

$$\frac{dI}{dt} = \beta S(t)I(t)$$

- Two state variables. One equation. Problem? No:

$$\frac{dS}{dt} = -\beta S(t)I(t)$$

- But $S(t) = N - I(t) \quad \Longrightarrow \quad I(t)$ is still the only variable:

Revised epidemic model: "SI model"

$$\frac{dI}{dt} = \beta S(t) I(t)$$

- Two state variables. One equation. Problem? No:

$$\frac{dS}{dt} = -\beta S(t) I(t)$$

- But $S(t) = N - I(t) \quad \Longrightarrow \quad I(t)$ is still the only variable:

$$\frac{dI}{dt} = \beta I(N - I)$$

Revised epidemic model: "SI model"

$$\frac{dI}{dt} = \beta S(t)I(t)$$

- Two state variables. One equation. Problem? No:

$$\frac{dS}{dt} = -\beta S(t)I(t)$$

- But $S(t) = N - I(t) \quad \Longrightarrow \quad I(t)$ is still the only variable:

$$\frac{dI}{dt} = \beta I(N - I)$$

- Is this a better model?

Revised epidemic model: “SI model”

$$\frac{dI}{dt} = \beta S(t)I(t)$$

- Two state variables. One equation. Problem? No:

$$\frac{dS}{dt} = -\beta S(t)I(t)$$

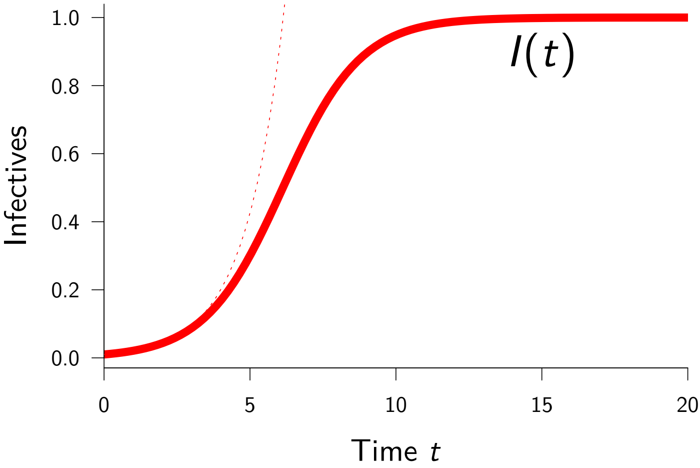
- But $S(t) = N - I(t) \quad \Longrightarrow \quad I(t)$ is still the only variable:

$$\frac{dI}{dt} = \beta I(N - I)$$

- Is this a better model?
- What does it predict?

SI model: Example solution

SI model: Example solution



SI model: Analysis $\frac{dI}{dt} = \beta I(N - I)$

SI model: Analysis $\frac{dI}{dt} = \beta I(N - I)$

- We can find the exact solution. How?

SI model: Analysis $\frac{dI}{dt} = \beta I(N - I)$

- We can find the exact solution. How? (Assignment 1)

SI model: Analysis $\frac{dI}{dt} = \beta I(N - I)$

- We can find the exact solution. How? (Assignment 1)

$$I(t) = \frac{I_0 e^{N\beta t}}{1 + (I_0/N)(e^{N\beta t} - 1)}$$

SI model: Analysis $\frac{dI}{dt} = \beta I(N - I)$

- We can find the exact solution. How? (Assignment 1)

$$I(t) = \frac{I_0 e^{N\beta t}}{1 + (I_0/N)(e^{N\beta t} - 1)}$$

- But exact solution is not particularly enlightening.

SI model: Analysis $\frac{dI}{dt} = \beta I(N - I)$

- We can find the exact solution. How? (Assignment 1)

$$I(t) = \frac{I_0 e^{N\beta t}}{1 + (I_0/N)(e^{N\beta t} - 1)}$$

- But exact solution is not particularly enlightening.
- Qualitative analysis:

SI model: Analysis $\frac{dI}{dt} = \beta I(N - I)$

- We can find the exact solution. How? (Assignment 1)

$$I(t) = \frac{I_0 e^{N\beta t}}{1 + (I_0/N)(e^{N\beta t} - 1)}$$

- But exact solution is not particularly enlightening.
- Qualitative analysis:
  - Initially $I \ll N$. What does the model predict then?

SI model: Analysis $\frac{dI}{dt} = \beta I(N - I)$

- We can find the exact solution. How? (Assignment 1)

$$I(t) = \frac{I_0 e^{N\beta t}}{1 + (I_0/N)(e^{N\beta t} - 1)}$$

- But exact solution is not particularly enlightening.

- Qualitative analysis:
    - Initially $I \ll N$. What does the model predict then? Exponential growth.

SI model: Analysis $\frac{dI}{dt} = \beta I(N - I)$

- We can find the exact solution. How? (Assignment 1)

$$I(t) = \frac{I_0 e^{N\beta t}}{1 + (I_0/N)(e^{N\beta t} - 1)}$$

- But exact solution is not particularly enlightening.

- Qualitative analysis:
  - Initially $I \ll N$. What does the model predict then? Exponential growth. Great!

SI model: Analysis $\frac{dI}{dt} = \beta I(N-I)$

- We can find the exact solution. How? (Assignment 1)

$$I(t) = \frac{I_0 e^{N\beta t}}{1 + (I_0/N)(e^{N\beta t} - 1)}$$

- But exact solution is not particularly enlightening.
- Qualitative analysis:
  - Initially $I \ll N$. What does the model predict then? Exponential growth. Great!
  - As I grows, growth rate slows. Why?

SI model: Analysis $\frac{dI}{dt} = \beta I(N - I)$

- We can find the exact solution. How? (Assignment 1)

$$I(t) = \frac{I_0 e^{N\beta t}}{1 + (I_0/N)(e^{N\beta t} - 1)}$$

- But exact solution is not particularly enlightening.

- Qualitative analysis:
  - Initially $I \ll N$. What does the model predict then?
    Exponential growth. Great!
  - As I grows, growth rate slows. Why?
    Fewer and fewer susceptibles to infect.

SI model: Analysis $\frac{dI}{dt} = \beta I(N - I)$

- We can find the exact solution. How? (Assignment 1)

$$I(t) = \frac{I_0 e^{N\beta t}}{1 + (I_0/N)(e^{N\beta t} - 1)}$$

- But exact solution is not particularly enlightening.
- Qualitative analysis:
  - Initially $I \ll N$. What does the model predict then? Exponential growth. Great!
  - As I grows, growth rate slows. Why? Fewer and fewer susceptibles to infect.
  - Asymptotic behaviour?

SI model: Analysis $\frac{dI}{dt} = \beta I(N - I)$

- We can find the exact solution. How? (Assignment 1)

$$I(t) = \frac{I_0 e^{N\beta t}}{1 + (I_0/N)(e^{N\beta t} - 1)}$$

- But exact solution is not particularly enlightening.

- Qualitative analysis:
  - Initially $I \ll N$. What does the model predict then?
    Exponential growth. Great!
  - As I grows, growth rate slows. Why?
    Fewer and fewer susceptibles to infect.
  - Asymptotic behaviour? Equilibria?

SI model: Analysis $\frac{dI}{dt} = \beta I(N - I)$

- We can find the exact solution. How? (Assignment 1)

$$I(t) = \frac{I_0 e^{N\beta t}}{1 + (I_0/N)(e^{N\beta t} - 1)}$$

- But exact solution is not particularly enlightening.

- Qualitative analysis:
  - Initially $I \ll N$. What does the model predict then?
    Exponential growth. Great!
  - As I grows, growth rate slows. Why?
    Fewer and fewer susceptibles to infect.
  - Asymptotic behaviour? Equilibria? Periodic orbits?

SI model: Analysis $\frac{dI}{dt} = \beta I(N - I)$

- We can find the exact solution. How? (Assignment 1)

$$I(t) = \frac{I_0 e^{N\beta t}}{1 + (I_0/N)(e^{N\beta t} - 1)}$$

- But exact solution is not particularly enlightening.
- Qualitative analysis:
  - Initially $I \ll N$. What does the model predict then? Exponential growth. Great!
  - As I grows, growth rate slows. Why? Fewer and fewer susceptibles to infect.
  - Asymptotic behaviour? Equilibria? Periodic orbits? (periodic orbit = recurrent epidemics)

SI model: Analysis $\quad \frac{dI}{dt} = \beta I(N - I)$

- We can find the exact solution. How? (Assignment 1)

$$I(t) = \frac{I_0 e^{N\beta t}}{1 + (I_0/N)(e^{N\beta t} - 1)}$$

- But exact solution is not particularly enlightening.

- Qualitative analysis:
  - Initially $I \ll N$. What does the model predict then? Exponential growth. Great!
  - As I grows, growth rate slows. Why? Fewer and fewer susceptibles to infect.
  - Asymptotic behaviour? Equilibria? Periodic orbits? (periodic orbit = recurrent epidemics)
  - (Non-trivial) periodic orbits impossible in one dimension (existence-uniqueness theorem).

SI model: Analysis $\quad \frac{dI}{dt} = \beta I(N - I)$

- We can find the exact solution. How? (Assignment 1)

$$I(t) = \frac{I_0 e^{N\beta t}}{1 + (I_0/N)(e^{N\beta t} - 1)}$$

- But exact solution is not particularly enlightening.
- Qualitative analysis:
  - Initially $I \ll N$. What does the model predict then? Exponential growth. Great!
  - As I grows, growth rate slows. Why? Fewer and fewer susceptibles to infect.
  - Asymptotic behaviour? Equilibria? Periodic orbits? (periodic orbit = recurrent epidemics)
  - (Non-trivial) periodic orbits impossible in one dimension (existence-uniqueness theorem).
  - Consider equilibria. . .

SI model: Equilibrium Analysis

$$\frac{dI}{dt} = \beta I(N - I)\,, \qquad I \in [0, N]$$

SI model: Equilibrium Analysis

$$\frac{dI}{dt} = \beta I(N - I)\,, \qquad I \in [0, N]$$

- Two equilibria:

SI model: Equilibrium Analysis

$$\frac{dI}{dt} = \beta I(N - I)\,, \qquad I \in [0, N]$$

- Two equilibria:
  - $I = 0$ Disease Free Equilibrium (**DFE**)

SI model: Equilibrium Analysis

$$\frac{dI}{dt} = \beta I(N - I) , \qquad I \in [0, N]$$

- Two equilibria:
  - $I = 0$ Disease Free Equilibrium (**DFE**)
  - $I = N$ Endemic Equilibrium (**EE**)

SI model: Equilibrium Analysis

$$\frac{dI}{dt} = \beta I(N - I)\,, \qquad I \in [0, N]$$

- Two equilibria:
  - $I = 0$ Disease Free Equilibrium (**DFE**)
  - $I = N$ Endemic Equilibrium (**EE**)
- Stability:

SI model: Equilibrium Analysis

$$\frac{dI}{dt} = \beta I(N - I)\,, \qquad I \in [0, N]$$

- Two equilibria:
  - $I = 0$ Disease Free Equilibrium (**DFE**)
  - $I = N$ Endemic Equilibrium (**EE**)
- Stability:
  - DFE is unstable

SI model: Equilibrium Analysis

$$\frac{dI}{dt} = \beta I(N - I)\,, \qquad I \in [0, N]$$

- Two equilibria:
  - $I = 0$ Disease Free Equilibrium (**DFE**)
  - $I = N$ Endemic Equilibrium (**EE**)
- Stability:
  - DFE is unstable ($0 < I < N \implies dI/dt > 0$)

SI model: Equilibrium Analysis

$$\frac{dI}{dt} = \beta I(N - I)\,, \qquad I \in [0, N]$$

- Two equilibria:
  - $I = 0$ Disease Free Equilibrium (**DFE**)
  - $I = N$ Endemic Equilibrium (**EE**)
- Stability:
  - DFE is unstable ($0 < I < N \implies dI/dt > 0$)
  - EE is locally asymptotically stable (**LAS**)

SI model: Equilibrium Analysis

$$\frac{dI}{dt} = \beta I(N - I)\,, \qquad I \in [0, N]$$

- Two equilibria:
  - $I = 0$ Disease Free Equilibrium (**DFE**)
  - $I = N$ Endemic Equilibrium (**EE**)
- Stability:
  - DFE is unstable ($0 < I < N \implies dI/dt > 0$)
  - EE is locally asymptotically stable (**LAS**)
  - EE is globally asymptotically stable (**GAS**)

SI model: Equilibrium Analysis

$$\frac{dI}{dt} = \beta I(N - I)\,, \qquad I \in [0, N]$$

- Two equilibria:
  - $I = 0$ Disease Free Equilibrium (**DFE**)
  - $I = N$ Endemic Equilibrium (**EE**)
- Stability:
  - DFE is unstable ($0 < I < N \implies dI/dt > 0$)
  - EE is locally asymptotically stable (**LAS**)
  - EE is globally asymptotically stable (**GAS**)
    (stability of EE follows from $0 < I < N \implies dI/dt > 0$)

SI model: Equilibrium Analysis

$$\frac{dI}{dt} = \beta I(N - I)\,, \qquad I \in [0, N]$$

- Two equilibria:
  - $I = 0$ Disease Free Equilibrium (**DFE**)
  - $I = N$ Endemic Equilibrium (**EE**)
- Stability:
  - DFE is unstable ($0 < I < N \implies dI/dt > 0$)
  - EE is locally asymptotically stable (**LAS**)
  - EE is globally asymptotically stable (**GAS**)
    (stability of EE follows from $0 < I < N \implies dI/dt > 0$)
    (GAS requires a little more analysis...

SI model: Equilibrium Analysis

$$\frac{dI}{dt} = \beta I(N - I)\,, \qquad I \in [0, N]$$

- Two equilibria:
  - $I = 0$ Disease Free Equilibrium (**DFE**)
  - $I = N$ Endemic Equilibrium (**EE**)
- Stability:
  - DFE is unstable ($0 < I < N \implies dI/dt > 0$)
  - EE is locally asymptotically stable (**LAS**)
  - EE is globally asymptotically stable (**GAS**)
    (stability of EE follows from $0 < I < N \implies dI/dt > 0$)
    (GAS requires a little more analysis... Assignment 1)

SI model: Equilibrium Analysis

$$\frac{dI}{dt} = \beta I(N - I) \,, \qquad I \in [0, N]$$

- Two equilibria:
  - $I = 0$ Disease Free Equilibrium (**DFE**)
  - $I = N$ Endemic Equilibrium (**EE**)
- Stability:
  - DFE is unstable ($0 < I < N \implies dI/dt > 0$)
  - EE is locally asymptotically stable (**LAS**)
  - EE is globally asymptotically stable (**GAS**)
    (stability of EE follows from $0 < I < N \implies dI/dt > 0$)
    (GAS requires a little more analysis... Assignment 1)
  - *Note:* In one dimension, global analysis always easy.

SI model: Equilibrium Analysis

$$\frac{dI}{dt} = \beta I(N - I)\,, \qquad I \in [0, N]$$

- Two equilibria:
  - $I = 0$ Disease Free Equilibrium (**DFE**)
  - $I = N$ Endemic Equilibrium (**EE**)
- Stability:
  - DFE is unstable ($0 < I < N \implies dI/dt > 0$)
  - EE is locally asymptotically stable (**LAS**)
  - EE is globally asymptotically stable (**GAS**)
    (stability of EE follows from $0 < I < N \implies dI/dt > 0$)
    (GAS requires a little more analysis... Assignment 1)
  - *Note:* In one dimension, global analysis always easy.
    In higher dimensions,

SI model: Equilibrium Analysis

$$\frac{dI}{dt} = \beta I(N - I)\,, \qquad I \in [0, N]$$

- Two equilibria:
  - $I = 0$ Disease Free Equilibrium (**DFE**)
  - $I = N$ Endemic Equilibrium (**EE**)
- Stability:
  - DFE is unstable ($0 < I < N \implies dI/dt > 0$)
  - EE is locally asymptotically stable (**LAS**)
  - EE is globally asymptotically stable (**GAS**)
    (stability of EE follows from $0 < I < N \implies dI/dt > 0$)
    (GAS requires a little more analysis... Assignment 1)
  - *Note:* In one dimension, global analysis always easy.
    In higher dimensions, often try to find Lyapunov function.

SI model: Equilibrium Analysis

$$\frac{dI}{dt} = \beta I(N - I)\,, \qquad I \in [0, N]$$

- Two equilibria:
  - $I = 0$ Disease Free Equilibrium (**DFE**)
  - $I = N$ Endemic Equilibrium (**EE**)
- Stability:
  - DFE is unstable ($0 < I < N \implies dI/dt > 0$)
  - EE is locally asymptotically stable (**LAS**)
  - EE is globally asymptotically stable (**GAS**)
    (stability of EE follows from $0 < I < N \implies dI/dt > 0$)
    (GAS requires a little more analysis... Assignment 1)
  - *Note:* In one dimension, global analysis always easy.
    In higher dimensions, often try to find Lyapunov function.
    (Lyapunov function for EE of SI model?...

SI model: Equilibrium Analysis

$$\frac{dI}{dt} = \beta I(N - I)\,, \qquad I \in [0, N]$$

- Two equilibria:
  - $I = 0$ Disease Free Equilibrium (**DFE**)
  - $I = N$ Endemic Equilibrium (**EE**)
- Stability:
  - DFE is unstable ($0 < I < N \implies dI/dt > 0$)
  - EE is locally asymptotically stable (**LAS**)
  - EE is globally asymptotically stable (**GAS**)
    (stability of EE follows from $0 < I < N \implies dI/dt > 0$)
    (GAS requires a little more analysis... Assignment 1)
  - *Note:* In one dimension, global analysis always easy.
    In higher dimensions, often try to find Lyapunov function.
    (Lyapunov function for EE of SI model?... Assignment 1)

SI model: Equilibrium Analysis

$$\frac{dI}{dt} = \beta I(N - I)\,, \qquad I \in [0, N]$$

- Two equilibria:
  - $I = 0$ Disease Free Equilibrium (**DFE**)
  - $I = N$ Endemic Equilibrium (**EE**)
- Stability:
  - DFE is unstable ($0 < I < N \implies dI/dt > 0$)
  - EE is locally asymptotically stable (**LAS**)
  - EE is globally asymptotically stable (**GAS**)
    (stability of EE follows from $0 < I < N \implies dI/dt > 0$)
    (GAS requires a little more analysis... Assignment 1)
  - *Note:* In one dimension, global analysis always easy.
    In higher dimensions, often try to find Lyapunov function.
    (Lyapunov function for EE of SI model?... Assignment 1)
- Conclusions identical for any $\beta > 0$.

SI model: Biological Inferences

SI model: Biological Inferences

- For *any* transmission rate β:

SI model: Biological Inferences

- For *any* transmission rate β:
  - Initially, exponential growth of cases.

SI model: Biological Inferences

- For *any* transmission rate β:
  - Initially, exponential growth of cases.
  - Eventually, convergence to equilibrium (EE) at which

SI model: Biological Inferences

- For *any* transmission rate β:
  - Initially, exponential growth of cases.
  - Eventually, convergence to equilibrium (EE) at which *everyone* in the population is infective.

SI model: Biological Inferences

- For *any* transmission rate β:
  - Initially, exponential growth of cases.
  - Eventually, convergence to equilibrium (EE) at which *everyone* in the population is infective. hmmm...

SI model: Biological Inferences

- For *any* transmission rate β:
  - Initially, exponential growth of cases.
  - Eventually, convergence to equilibrium (EE) at which *everyone* in the population is infective. hmmm...
- Is this model better than our first naïve model?

SI model: Biological Inferences

- For *any* transmission rate β:
  - Initially, exponential growth of cases.
  - Eventually, convergence to equilibrium (EE) at which *everyone* in the population is infective. hmmm...
- Is this model better than our first naïve model?
  YES.

SI model: Biological Inferences

- For *any* transmission rate β:
  - Initially, exponential growth of cases.
  - Eventually, convergence to equilibrium (EE) at which *everyone* in the population is infective. hmmm...
- Is this model better than our first naïve model?
  YES.
  - Still correctly predict initial exponential growth.

SI model: Biological Inferences

- For *any* transmission rate β:
  - Initially, exponential growth of cases.
  - Eventually, convergence to equilibrium (EE) at which *everyone* in the population is infective. hmmm...
- Is this model better than our first naïve model? YES.
  - Still correctly predict initial exponential growth.
  - Can match epidemic curve for longer

SI model: Biological Inferences

- For *any* transmission rate β:
  - Initially, exponential growth of cases.
  - Eventually, convergence to equilibrium (EE) at which *everyone* in the population is infective. hmmm...
- Is this model better than our first naïve model?
  YES.
  - Still correctly predict initial exponential growth.
  - Can match epidemic curve for longer (up to the peak).

SI model: Biological Inferences

- For *any* transmission rate β:
  - Initially, exponential growth of cases.
  - Eventually, convergence to equilibrium (EE) at which *everyone* in the population is infective. hmmm. . .
- Is this model better than our first naïve model? YES.
  - Still correctly predict initial exponential growth.
  - Can match epidemic curve for longer (up to the peak).
  - Does not predict absurd unbounded growth in infective population.

SI model: Biological Inferences

- For *any* transmission rate β:
  - Initially, exponential growth of cases.
  - Eventually, convergence to equilibrium (EE) at which *everyone* in the population is infective. hmmm...
- Is this model better than our first naïve model?
  YES.
  - Still correctly predict initial exponential growth.
  - Can match epidemic curve for longer (up to the peak).
  - Does not predict absurd unbounded growth in infective population.
  - But this model cannot explain the decline of the epidemic.

SI model: Biological Inferences

- For *any* transmission rate β:
  - Initially, exponential growth of cases.
  - Eventually, convergence to equilibrium (EE) at which *everyone* in the population is infective. hmmm...
- Is this model better than our first naïve model?
  YES.
  - Still correctly predict initial exponential growth.
  - Can match epidemic curve for longer (up to the peak).
  - Does not predict absurd unbounded growth in infective population.
  - But this model cannot explain the decline of the epidemic.
- What should we do?

SI model: Biological Inferences

- For *any* transmission rate β:
  - Initially, exponential growth of cases.
  - Eventually, convergence to equilibrium (EE) at which *everyone* in the population is infective. hmmm. . .
- Is this model better than our first naïve model?
  YES.
  - Still correctly predict initial exponential growth.
  - Can match epidemic curve for longer (up to the peak).
  - Does not predict absurd unbounded growth in infective population.
  - But this model cannot explain the decline of the epidemic.
- What should we do? Two obvious options:

SI model: Biological Inferences

- For *any* transmission rate β:
  - Initially, exponential growth of cases.
  - Eventually, convergence to equilibrium (EE) at which *everyone* in the population is infective. hmmm...
- Is this model better than our first naïve model?
  YES.
  - Still correctly predict initial exponential growth.
  - Can match epidemic curve for longer (up to the peak).
  - Does not predict absurd unbounded growth in infective population.
  - But this model cannot explain the decline of the epidemic.
- What should we do? Two obvious options:
  1. Get depressed, drop the course.

SI model: Biological Inferences

- For *any* transmission rate β:
  - Initially, exponential growth of cases.
  - Eventually, convergence to equilibrium (EE) at which *everyone* in the population is infective. hmmm...
- Is this model better than our first naïve model? YES.
  - Still correctly predict initial exponential growth.
  - Can match epidemic curve for longer (up to the peak).
  - Does not predict absurd unbounded growth in infective population.
  - But this model cannot explain the decline of the epidemic.
- What should we do? Two obvious options:
  1. Get depressed, drop the course.
  2. Try to improve the model.

Recall motivating data: 1918 flu in Philadelphia

Recall motivating data: 1918 flu in Philadelphia

- Mortality curve (linear scale)

Recall motivating data: 1918 flu in Philadelphia

- Mortality curve (linear scale)
- Mortality curve (log scale)

How can we improve on the SI model?

How can we improve on the SI model?

- Include a key biological fact:

How can we improve on the SI model?

- Include a key biological fact:
  Individuals do not stay infectious with flu forever

How can we improve on the SI model?

- Include a key biological fact:
  Individuals do not stay infectious with flu forever
- Either they *recover* and are immune thereafter, or they *die*

How can we improve on the SI model?

- Include a key biological fact:
  Individuals do not stay infectious with flu forever
- Either they *recover* and are immune thereafter, or they *die* (doesn't matter which)

How can we improve on the SI model?

- Include a key biological fact:
  Individuals do not stay infectious with flu forever
- Either they *recover* and are immune thereafter, or they *die* (doesn't matter which) (well, maybe to them it does)

How can we improve on the SI model?

- Include a key biological fact:
  Individuals do not stay infectious with flu forever
- Either they *recover* and are immune thereafter, or they *die* (doesn't matter which) (well, maybe to them it does)
- Why don't we care if someone recovers or dies?

How can we improve on the SI model?

- Include a key biological fact:
  Individuals do not stay infectious with flu forever
- Either they *recover* and are immune thereafter, or they *die* (doesn't matter which) (well, maybe to them it does)
- Why don't we care if someone recovers or dies?
  (*i.e.*, Why doesn't it affect our dynamical inferences?)

How can we improve on the SI model?

- Include a key biological fact:
  Individuals do not stay infectious with flu forever
- Either they *recover* and are immune thereafter, or they *die* (doesn't matter which) (well, maybe to them it does)
- Why don't we care if someone recovers or dies?
  (*i.e.*, Why doesn't it affect our dynamical inferences?)
- Because in either case the individual is *removed* from the transmission process, hence

How can we improve on the SI model?

- Include a key biological fact:
  Individuals do not stay infectious with flu forever
- Either they *recover* and are immune thereafter, or they *die* (doesn't matter which) (well, maybe to them it does)
- Why don't we care if someone recovers or dies?
  (*i.e.*, Why doesn't it affect our dynamical inferences?)
- Because in either case the individual is *removed* from the transmission process, hence cannot affect the future pattern of the epidemic.

The SIR model

The SIR model

Introduce new class of removed individuals:

The SIR model

Introduce new class of removed individuals:

- $R(t) =$ number of individuals who have either recovered and are now immune or have died

The SIR model

Introduce new class of removed individuals:

- $R(t) =$ number of individuals who have either recovered and are now immune or have died
- Let $\gamma =$ rate of removal from the infective class (via recovery or death)

The SIR model

Introduce new class of removed individuals:

- $R(t) =$ number of individuals who have either recovered and are now immune or have died
- Let $\gamma =$ rate of removal from the infective class (via recovery or death)

$$\frac{dS}{dt} = -\beta SI$$
$$\frac{dI}{dt} = \beta SI - \gamma I$$
$$\frac{dR}{dt} = \gamma I$$

The SIR model

Introduce new class of removed individuals:

- $R(t)$ = number of individuals who have either recovered and are now immune or have died
- Let γ = rate of removal from the infective class (via recovery or death)

$$\frac{dS}{dt} = -\beta S I$$
$$\frac{dI}{dt} = \beta S I - \gamma I$$
$$\frac{dR}{dt} = \gamma I$$

- Note: $\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0 \implies S + I + R = N = \text{constant}$

The SIR model

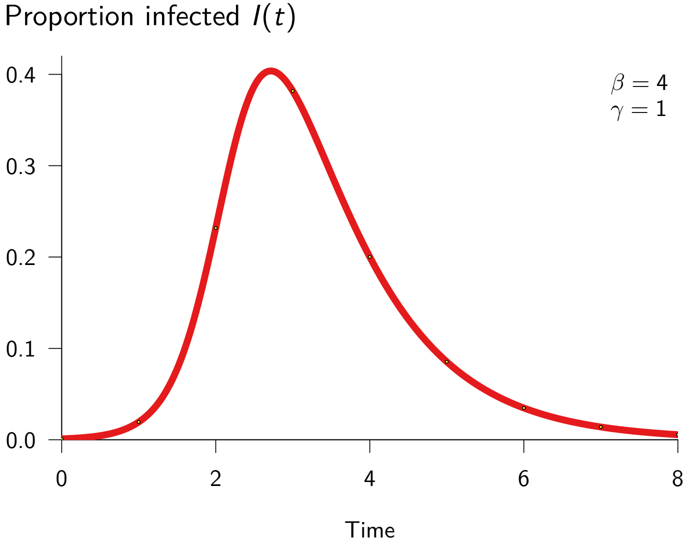
Introduce new class of removed individuals:

- $R(t) =$ number of individuals who have either recovered and are now immune or have died
- Let $\gamma =$ rate of removal from the infective class (via recovery or death)

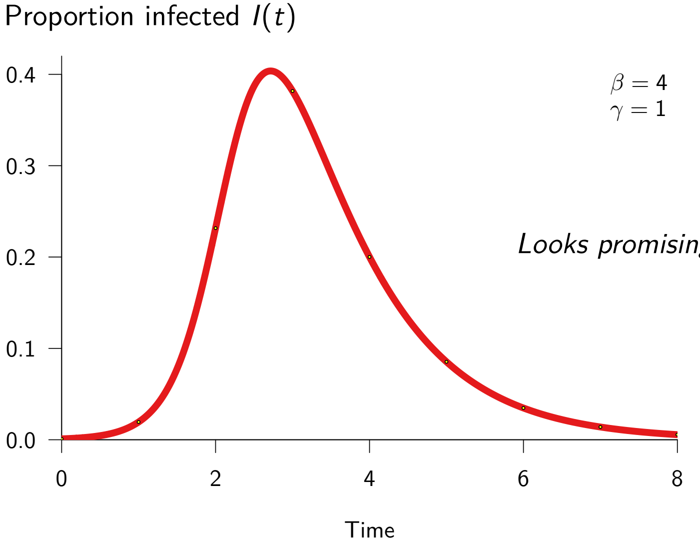
$$\frac{dS}{dt} = -\beta S I$$
$$\frac{dI}{dt} = \beta S I - \gamma I$$
$$\frac{dR}{dt} = \gamma I$$

- Note: $\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0 \implies S + I + R = N =$ constant
- Convenient to rescale variables by N and interpret S, I, R as *proportions* of the population in each disease state.

The SIR model: Example numerical solution

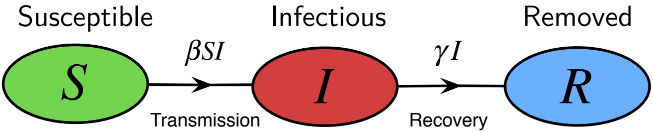


The SIR model: Example numerical solution



Looks promising...

The SIR model: Flow Chart and Parameters

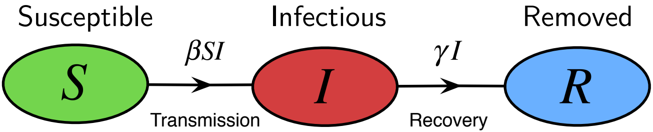


$$\frac{dS}{dt} = -\beta S I$$

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

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

The SIR model: Flow Chart and Parameters



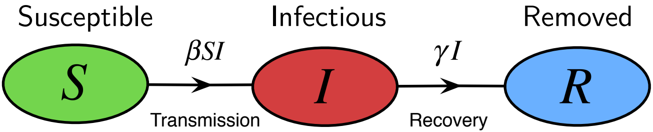
$$\frac{dS}{dt} = -\beta S I$$

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

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

- **Parameters:**

The SIR model: Flow Chart and Parameters



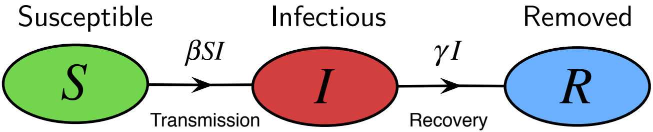
$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

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

- **Parameters:**
  - Transmission rate β

The SIR model: Flow Chart and Parameters



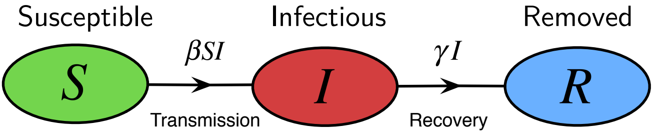
$$\frac{dS}{dt} = -\beta S I$$

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

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

- **Parameters:**
  - Transmission rate β
  - Recovery rate γ

The SIR model: Flow Chart and Parameters



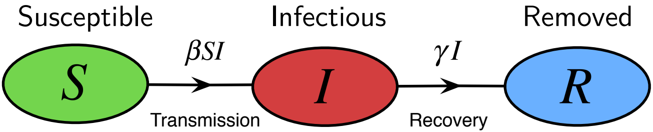
$$\frac{dS}{dt} = -\beta S I$$

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

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

- **Parameters:**
  - Transmission rate β
  - Recovery rate γ (or Removal rate)

The SIR model: Derived Parameters

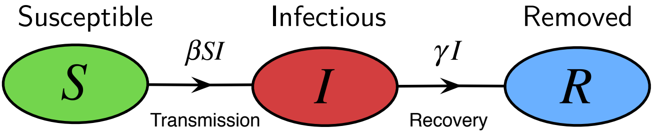


$$\frac{dS}{dt} = -\beta S I$$

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

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

The SIR model: Derived Parameters



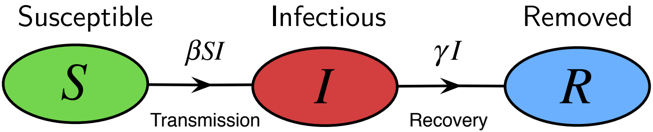
$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

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

- **Derived Parameters:**

The SIR model: Derived Parameters



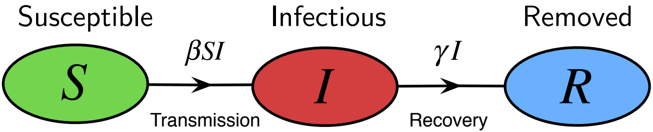
$$\frac{dS}{dt} = -\beta S I$$

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

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

- **Derived Parameters:**
  - Initial growth rate $\quad \beta - \gamma$

The SIR model: Derived Parameters



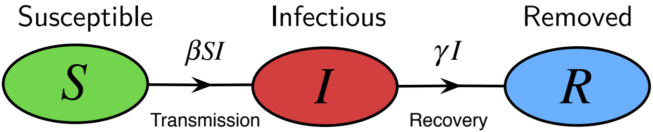
$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

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

- **Derived Parameters:**
  - Initial growth rate $\beta - \gamma$
  - Mean infectious period $\frac{1}{\gamma}$

The SIR model: Derived Parameters



$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

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

- **Derived Parameters:**
  - Initial growth rate $\beta - \gamma$
  - Mean infectious period $\frac{1}{\gamma}$
  - Basic Reproduction Number

$$\mathcal{R}_0 = \frac{\beta}{\gamma}$$

The SIR model: Derived parameters

The initial growth rate

The SIR model: Derived parameters

The initial growth rate

- How do we calculate the initial growth rate from our model?

The SIR model: Derived parameters

The initial growth rate

- How do we calculate the initial growth rate from our model?
- Consider change in prevalence initially (when $I \ll 1$):

The SIR model: Derived parameters

The initial growth rate

- How do we calculate the initial growth rate from our model?
- Consider change in prevalence initially (when $I \ll 1$):

$$\frac{dI}{dt} =$$

The SIR model: Derived parameters

The initial growth rate

- How do we calculate the initial growth rate from our model?
- Consider change in prevalence initially (when $I \ll 1$):

$$\frac{dI}{dt} = \beta SI - \gamma I$$

The SIR model: Derived parameters

The initial growth rate

- How do we calculate the initial growth rate from our model?
- Consider change in prevalence initially (when $I \ll 1$):

$$\begin{aligned} \frac{dI}{dt} &= \beta SI - \gamma I \\ &= \end{aligned}$$

The SIR model: Derived parameters

The initial growth rate

- How do we calculate the initial growth rate from our model?
- Consider change in prevalence initially (when $I \ll 1$):

$$\begin{aligned}\frac{dI}{dt} &= \beta S I - \gamma I \\ &= \big(\beta S - \gamma\big) I\end{aligned}$$

The SIR model: Derived parameters

The initial growth rate

- How do we calculate the initial growth rate from our model?
- Consider change in prevalence initially (when $I \ll 1$):

$$\begin{aligned} \frac{dI}{dt} &= \beta SI - \gamma I \\ &= \Big(\beta S - \gamma\Big) I \\ &\approx \end{aligned}$$

The SIR model: Derived parameters

The initial growth rate

- How do we calculate the initial growth rate from our model?
- Consider change in prevalence initially (when $I \ll 1$):

$$\begin{aligned}\frac{dI}{dt} &= \beta SI - \gamma I \\ &= \Big(\beta S - \gamma\Big) I \\ &\approx \Big(\beta - \gamma\Big) I\end{aligned}$$

The SIR model: Derived parameters

The initial growth rate

- How do we calculate the initial growth rate from our model?
- Consider change in prevalence initially (when $I \ll 1$):

$$\begin{aligned} \frac{dI}{dt} &= \beta SI - \gamma I \\ &= \Big(\beta S - \gamma\Big) I \\ &\approx \Big(\beta - \gamma\Big) I \qquad \text{if } S \sim 1 \text{ initially.} \end{aligned}$$

The SIR model: Derived parameters

The initial growth rate

- How do we calculate the initial growth rate from our model?
- Consider change in prevalence initially (when $I \ll 1$):

$$\begin{aligned} \frac{dI}{dt} &= \beta SI - \gamma I \\ &= \Big(\beta S - \gamma\Big) I \\ &\approx \Big(\beta - \gamma\Big) I \qquad \text{if } S \sim 1 \text{ initially.} \end{aligned}$$

- $\therefore$ Initially $\quad I(t) \approx I_0 e^{(\beta-\gamma)t}$.

The SIR model: Derived parameters

The initial growth rate

- How do we calculate the initial growth rate from our model?
- Consider change in prevalence initially (when $I \ll 1$):

$$\begin{aligned}\frac{dI}{dt} &= \beta SI - \gamma I \\ &= \Big(\beta S - \gamma\Big) I \\ &\approx \Big(\beta - \gamma\Big) I \qquad \text{if } S \sim 1 \text{ initially.}\end{aligned}$$

- $\therefore$ Initially $\quad I(t) \approx I_0 e^{(\beta-\gamma)t}$.
- $\therefore$ Initial slope of logged prevalence curve is $\quad \beta - \gamma$.

The SIR model: Derived parameters

The mean infectious period

The SIR model: Derived parameters

The mean infectious period

- How do we calculate the mean infectious period from our model?

The SIR model: Derived parameters

The mean infectious period

- How do we calculate the mean infectious period from our model?
- Suppose at time 0 there are I_0 infectious individuals, and suppose we can *prevent contact with susceptibles*.

The SIR model: Derived parameters

The mean infectious period

- How do we calculate the mean infectious period from our model?
- Suppose at time 0 there are I_0 infectious individuals, and suppose we can *prevent contact with susceptibles*.
- The equation for I then simplifies to

The SIR model: Derived parameters

The mean infectious period

- How do we calculate the mean infectious period from our model?
- Suppose at time 0 there are I_0 infectious individuals, and suppose we can *prevent contact with susceptibles*.
- The equation for I then simplifies to

$$\frac{dI}{dt} = -\gamma I\,, \qquad I(0) = I_0$$

The SIR model: Derived parameters

The mean infectious period

- How do we calculate the mean infectious period from our model?
- Suppose at time 0 there are I_0 infectious individuals, and suppose we can *prevent contact with susceptibles*.
- The equation for I then simplifies to

$$\frac{dI}{dt} = -\gamma I\,, \qquad I(0) = I_0$$

- We can solve this immediately to find

$$I(t) =$$

The SIR model: Derived parameters

The mean infectious period

- How do we calculate the mean infectious period from our model?
- Suppose at time 0 there are I_0 infectious individuals, and suppose we can *prevent contact with susceptibles*.
- The equation for I then simplifies to

$$\frac{dI}{dt} = -\gamma I\,, \qquad I(0) = I_0$$

- We can solve this immediately to find

$$I(t) = I_0 e^{-\gamma t}$$

The SIR model: Derived parameters

The mean infectious period, continued. . .

The SIR model: Derived parameters

The mean infectious period, continued. . .

- Thus, after time t, the number of people still infectious is reduced by a factor

The SIR model: Derived parameters

The mean infectious period, continued...

- Thus, after time t, the number of people still infectious is reduced by a factor $e^{-\gamma t}$

The SIR model: Derived parameters

The mean infectious period, continued...

- Thus, after time t, the number of people still infectious is reduced by a factor $e^{-\gamma t}$
- *i.e.*, the proportion of individuals who have an infectious period shorter than t is

The SIR model: Derived parameters

The mean infectious period, continued...

- Thus, after time t, the number of people still infectious is reduced by a factor $e^{-\gamma t}$
- *i.e.*, the proportion of individuals who have an infectious period shorter than t is $1 - e^{-\gamma t}$

The SIR model: Derived parameters

The mean infectious period, continued...

- Thus, after time t, the number of people still infectious is reduced by a factor $e^{-\gamma t}$
- *i.e.*, the proportion of individuals who have an infectious period shorter than t is $1 - e^{-\gamma t}$
- *i.e.*, the cumulative distribution of the infectious period is $C(t) = 1 - e^{-\gamma t}$.

The SIR model: Derived parameters

The mean infectious period, continued...

- Thus, after time t, the number of people still infectious is reduced by a factor $e^{-\gamma t}$
- *i.e.*, the proportion of individuals who have an infectious period shorter than t is $1 - e^{-\gamma t}$
- *i.e.*, the cumulative distribution of the infectious period is $C(t) = 1 - e^{-\gamma t}$.
- Therefore, the probability density of the infectious period is $p(t) =$

The SIR model: Derived parameters

The mean infectious period, continued. . .

- Thus, after time t, the number of people still infectious is reduced by a factor $e^{-\gamma t}$
- *i.e.*, the proportion of individuals who have an infectious period shorter than t is $1 - e^{-\gamma t}$
- *i.e.*, the cumulative distribution of the infectious period is $C(t) = 1 - e^{-\gamma t}$.
- Therefore, the probability density of the infectious period is $p(t) = C'(t) =$

The SIR model: Derived parameters

The mean infectious period, continued...

- Thus, after time t, the number of people still infectious is reduced by a factor $e^{-\gamma t}$
- *i.e.*, the proportion of individuals who have an infectious period shorter than t is $1 - e^{-\gamma t}$
- *i.e.*, the cumulative distribution of the infectious period is $C(t) = 1 - e^{-\gamma t}$.
- Therefore, the probability density of the infectious period is $p(t) = C'(t) = \frac{d}{dt}\left(1 - e^{-\gamma t}\right) = \gamma e^{-\gamma t}$

The SIR model: Derived parameters

The mean infectious period, continued...

- Thus, after time t, the number of people still infectious is reduced by a factor $e^{-\gamma t}$
- *i.e.*, the proportion of individuals who have an infectious period shorter than t is $1 - e^{-\gamma t}$
- *i.e.*, the cumulative distribution of the infectious period is $C(t) = 1 - e^{-\gamma t}$.
- Therefore, the probability density of the infectious period is $p(t) = C'(t) = \frac{d}{dt}\left(1 - e^{-\gamma t}\right) = \gamma e^{-\gamma t}$
- The mean of this exponential probability distribution is

The SIR model: Derived parameters

The mean infectious period, continued...

- Thus, after time t, the number of people still infectious is reduced by a factor $e^{-\gamma t}$
- *i.e.*, the proportion of individuals who have an infectious period shorter than t is $1 - e^{-\gamma t}$
- *i.e.*, the cumulative distribution of the infectious period is $C(t) = 1 - e^{-\gamma t}$.
- Therefore, the probability density of the infectious period is $p(t) = C'(t) = \frac{d}{dt}\Big(1 - e^{-\gamma t}\Big) = \gamma e^{-\gamma t}$
- The mean of this exponential probability distribution is $\int_0^\infty t\,p(t)\,dt =$

The SIR model: Derived parameters

The mean infectious period, continued...

- Thus, after time t, the number of people still infectious is reduced by a factor $e^{-\gamma t}$
- *i.e.*, the proportion of individuals who have an infectious period shorter than t is $1 - e^{-\gamma t}$
- *i.e.*, the cumulative distribution of the infectious period is $C(t) = 1 - e^{-\gamma t}$.
- Therefore, the probability density of the infectious period is $p(t) = C'(t) = \frac{d}{dt}\left(1 - e^{-\gamma t}\right) = \gamma e^{-\gamma t}$
- The mean of this exponential probability distribution is $\int_0^\infty t\, p(t)\, dt = \int_0^\infty t\, \gamma e^{-\gamma t}\, dt =$

The SIR model: Derived parameters

The mean infectious period, continued...

- Thus, after time t, the number of people still infectious is reduced by a factor $e^{-\gamma t}$
- *i.e.*, the proportion of individuals who have an infectious period shorter than t is $1 - e^{-\gamma t}$
- *i.e.*, the cumulative distribution of the infectious period is $C(t) = 1 - e^{-\gamma t}$.
- Therefore, the probability density of the infectious period is $p(t) = C'(t) = \frac{d}{dt}\left(1 - e^{-\gamma t}\right) = \gamma e^{-\gamma t}$
- The mean of this exponential probability distribution is $\int_0^\infty t\, p(t)\, dt = \int_0^\infty t\, \gamma e^{-\gamma t}\, dt = \frac{1}{\gamma}$

The SIR model: Derived parameters

The basic reproduction number $\mathcal{R}_0$

$$\begin{aligned}\mathcal{R}_0 &= \beta \cdot \frac{1}{\gamma} \\ &= \end{aligned}$$

The SIR model: Derived parameters

The basic reproduction number $\mathcal{R}_0$

$$\begin{aligned}\mathcal{R}_0 &= \beta \cdot \frac{1}{\gamma} \\ &= (\text{transmission rate})\end{aligned}$$

The SIR model: Derived parameters

The basic reproduction number $\mathcal{R}_0$

$$\begin{aligned}\mathcal{R}_0 &= \beta \cdot \frac{1}{\gamma} \\ &= (\text{transmission rate}) \\ &\qquad\qquad \times (\text{mean infectious period})\end{aligned}$$

The SIR model: Derived parameters

The basic reproduction number $\mathcal{R}_0$

$$\mathcal{R}_0 = \beta \cdot \frac{1}{\gamma}$$
$$= (\text{transmission rate}) \times (\text{mean infectious period})$$

- $\mathcal{R}_0$ is dimensionless

The SIR model: Derived parameters

The basic reproduction number $\mathcal{R}_0$

$$\begin{aligned}\mathcal{R}_0 &= \beta \cdot \frac{1}{\gamma} \\ &= (\text{transmission rate}) \\ &\qquad\qquad \times (\text{mean infectious period})\end{aligned}$$

- $\mathcal{R}_0$ is dimensionless
- $\mathcal{R}_0$ is the average number of secondary cases caused by a primary case (in a wholly susceptible population).

The SIR model: Derived parameters

The basic reproduction number $\mathcal{R}_0$

$$\begin{aligned}\mathcal{R}_0 &= \beta \cdot \frac{1}{\gamma} \\ &= (\text{transmission rate}) \\ &\qquad\qquad \times (\text{mean infectious period})\end{aligned}$$

- $\mathcal{R}_0$ is dimensionless
- $\mathcal{R}_0$ is the average number of secondary cases caused by a primary case (in a wholly susceptible population).
- We must have $\mathcal{R}_0 > 1$ to have an epidemic. Why?

The SIR model: Derived parameters

The basic reproduction number $\mathcal{R}_0$

$$\begin{aligned}\mathcal{R}_0 &= \beta \cdot \frac{1}{\gamma} \\ &= (\text{transmission rate}) \\ &\qquad \times (\text{mean infectious period})\end{aligned}$$

- $\mathcal{R}_0$ is dimensionless
- $\mathcal{R}_0$ is the average number of secondary cases caused by a primary case (in a wholly susceptible population).
- We must have $\mathcal{R}_0 > 1$ to have an epidemic. Why?
  - $\frac{dI}{dt} = \beta SI - \gamma I = \big(\mathcal{R}_0 S - 1\big)\gamma I$

The SIR model: Derived parameters

The basic reproduction number $\mathcal{R}_0$

$$\begin{aligned}\mathcal{R}_0 &= \beta \cdot \frac{1}{\gamma} \\ &= (\text{transmission rate}) \\ &\qquad \times (\text{mean infectious period})\end{aligned}$$

- $\mathcal{R}_0$ is dimensionless
- $\mathcal{R}_0$ is the average number of secondary cases caused by a primary case (in a wholly susceptible population).
- We must have $\mathcal{R}_0 > 1$ to have an epidemic. Why?
  - $\frac{dI}{dt} = \beta SI - \gamma I = \big(\mathcal{R}_0 S - 1\big)\gamma I$
  - $\therefore \mathcal{R}_0 \le 1 \implies$

The SIR model: Derived parameters

The basic reproduction number $\mathcal{R}_0$

$$\begin{aligned}\mathcal{R}_0 &= \beta \cdot \frac{1}{\gamma} \\ &= (\text{transmission rate}) \\ &\qquad\qquad \times (\text{mean infectious period})\end{aligned}$$

- $\mathcal{R}_0$ is dimensionless
- $\mathcal{R}_0$ is the average number of secondary cases caused by a primary case (in a wholly susceptible population).
- We must have $\mathcal{R}_0 > 1$ to have an epidemic. Why?
  - $\frac{dI}{dt} = \beta SI - \gamma I = \big(\mathcal{R}_0 S - 1\big)\gamma I$
  - $\therefore \mathcal{R}_0 \le 1 \implies \frac{dI}{dt} \le 0$ for all $(S, I) \in [0,1]^2 \implies$

The SIR model: Derived parameters

The basic reproduction number $\mathcal{R}_0$

$$\begin{aligned}\mathcal{R}_0 &= \beta \cdot \frac{1}{\gamma} \\ &= \text{(transmission rate)} \\ &\qquad \times \text{(mean infectious period)}\end{aligned}$$

- $\mathcal{R}_0$ is dimensionless
- $\mathcal{R}_0$ is the average number of secondary cases caused by a primary case (in a wholly susceptible population).
- We must have $\mathcal{R}_0 > 1$ to have an epidemic. Why?
  - $\frac{dI}{dt} = \beta SI - \gamma I = \big(\mathcal{R}_0 S - 1\big)\gamma I$
  - $\therefore \mathcal{R}_0 \le 1 \implies \frac{dI}{dt} \le 0$ for all $(S, I) \in [0,1]^2 \implies$ no growth

The SIR model: Analysis (biological well-posedness)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Be careful:
Is this a sensible biological model?

The SIR model: Analysis (biological well-posedness)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- We need S, I and R all non-negative at all times.

Be careful:
Is this a sensible biological model?

The SIR model: Analysis (biological well-posedness)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- We need S, I and R all non-negative at all times.
- Does $0 \leq S(0) + I(0) \leq 1$ imply $0 \leq S(t) + I(t) \leq 1$ for all $t > 0$?

Be careful:
Is this a sensible biological model?

The SIR model: Analysis (biological well-posedness)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- We need S, I and R all non-negative at all times.
- Does $0 \leq S(0) + I(0) \leq 1$ imply $0 \leq S(t) + I(t) \leq 1$ for all $t > 0$?
  - $S = 0 \implies S' = 0$, so

Be careful:
Is this a sensible biological model?

The SIR model: Analysis (biological well-posedness)

$$\frac{dS}{dt} = -\beta S I$$

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

- We need S, I and R all non-negative at all times.
- Does $0 \leq S(0) + I(0) \leq 1$ imply $0 \leq S(t) + I(t) \leq 1$ for all $t > 0$?
  - $S = 0 \implies S' = 0$, so $S(0) \geq 0 \implies S(t) \geq 0 \; \forall t > 0$.

Be careful:
Is this a sensible biological model?

The SIR model: Analysis (biological well-posedness)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Be careful:
Is this a sensible biological model?

- We need S, I and R all non-negative at all times.
- Does $0 \leq S(0) + I(0) \leq 1$ imply $0 \leq S(t) + I(t) \leq 1$ for all $t > 0$?
  - $S = 0 \implies S' = 0$, so $S(0) \geq 0 \implies S(t) \geq 0 \; \forall t > 0$.
  - $I = 0 \implies I' = 0$, so

The SIR model: Analysis (biological well-posedness)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Be careful:
Is this a sensible biological model?

- We need S, I and R all non-negative at all times.
- Does $0 \le S(0) + I(0) \le 1$ imply $0 \le S(t) + I(t) \le 1$ for all $t > 0$?
  - $S = 0 \implies S' = 0$, so $S(0) \ge 0 \implies S(t) \ge 0 \ \forall t > 0$.
  - $I = 0 \implies I' = 0$, so $I(0) \ge 0 \implies I(t) \ge 0 \ \forall t > 0$.

The SIR model: Analysis (biological well-posedness)

$$\frac{dS}{dt} = -\beta S I$$

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

Be careful:
Is this a sensible biological model?

- We need S, I and R all non-negative at all times.
- Does $0 \leq S(0) + I(0) \leq 1$ imply $0 \leq S(t) + I(t) \leq 1$ for all $t > 0$?
  - $S = 0 \implies S' = 0$, so $S(0) \geq 0 \implies S(t) \geq 0 \ \forall t > 0$.
  - $I = 0 \implies I' = 0$, so $I(0) \geq 0 \implies I(t) \geq 0 \ \forall t > 0$.
  - $(S + I)' = S' + I' = -\gamma I \leq 0$ $\implies$

The SIR model: Analysis (biological well-posedness)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Be careful:
Is this a sensible biological model?

- We need S, I and R all non-negative at all times.
- Does $0 \leq S(0) + I(0) \leq 1$ imply $0 \leq S(t) + I(t) \leq 1$ for all $t > 0$?
  - $S = 0 \implies S' = 0$, so $S(0) \geq 0 \implies S(t) \geq 0 \; \forall t > 0$.
  - $I = 0 \implies I' = 0$, so $I(0) \geq 0 \implies I(t) \geq 0 \; \forall t > 0$.
  - $(S + I)' = S' + I' = -\gamma I \leq 0$
    $\implies S + I$ is always non-increasing
    $\implies$

The SIR model: Analysis (biological well-posedness)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Be careful:
Is this a sensible biological model?

- We need S, I and R all non-negative at all times.
- Does $0 \le S(0) + I(0) \le 1$ imply $0 \le S(t) + I(t) \le 1$ for all $t > 0$?
  - $S = 0 \implies S' = 0$, so $S(0) \ge 0 \implies S(t) \ge 0 \; \forall t > 0$.
  - $I = 0 \implies I' = 0$, so $I(0) \ge 0 \implies I(t) \ge 0 \; \forall t > 0$.
  - $(S + I)' = S' + I' = -\gamma I \le 0$
    $\implies S + I$ is always non-increasing
    $\implies S(t) + I(t) \le S(0) + I(0) \le 1$.

The SIR model: Analysis (equilibria *etc.*)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

The SIR model: Analysis (equilibria *etc.*)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- **Equilibria:**

The SIR model: Analysis (equilibria *etc.*)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- **Equilibria:**
  $(S, I) = (S_0, 0)$ for any $S_0 \in [0, 1]$

The SIR model: Analysis (equilibria *etc.*)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- **Equilibria:**
  $(S, I) = (S_0, 0)$ for any $S_0 \in [0, 1]$
  - Continuum of equilibria.

The SIR model: Analysis (equilibria *etc.*)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- **Equilibria:** $(S, I) = (S_0, 0)$ for any $S_0 \in [0, 1]$
  - Continuum of equilibria.
  - Does this make sense?

The SIR model: Analysis (equilibria *etc.*)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- **Equilibria:**
  $(S, I) = (S_0, 0)$ for any $S_0 \in [0, 1]$
  - Continuum of equilibria.
  - Does this make sense?
  - Biological meaning of equilibria?

The SIR model: Analysis (equilibria *etc.*)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- **Equilibria:**
  $(S, I) = (S_0, 0)$ for any $S_0 \in [0, 1]$
  - Continuum of equilibria.
  - Does this make sense?
  - Biological meaning of equilibria?

- **Linearization:**

The SIR model: Analysis (equilibria *etc.*)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- **Equilibria:** $(S, I) = (S_0, 0)$ for any $S_0 \in [0, 1]$
  - Continuum of equilibria.
  - Does this make sense?
  - Biological meaning of equilibria?

- **Linearization:**
  - $DF_{(S,I)} = \begin{pmatrix} -\beta I & -\beta S \\ \beta I & \beta S - \gamma \end{pmatrix}$

The SIR model: Analysis (equilibria *etc.*)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- **Equilibria:** $(S, I) = (S_0, 0)$ for any $S_0 \in [0, 1]$
  - Continuum of equilibria.
  - Does this make sense?
  - Biological meaning of equilibria?

- **Linearization:**
  - $DF_{(S,I)} = \begin{pmatrix} -\beta I & -\beta S \\ \beta I & \beta S - \gamma \end{pmatrix}$
  - $DF_{(S_0,0)} = \begin{pmatrix} 0 & -\beta S_0 \\ 0 & \beta S_0 - \gamma \end{pmatrix}$

The SIR model: Analysis (equilibria *etc.*)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- **Equilibria:**
  $(S, I) = (S_0, 0)$ for any $S_0 \in [0, 1]$
  - Continuum of equilibria.
  - Does this make sense?
  - Biological meaning of equilibria?

- **Linearization:**
  - $DF_{(S,I)} = \begin{pmatrix} -\beta I & -\beta S \\ \beta I & \beta S - \gamma \end{pmatrix}$
  - $DF_{(S_0,0)} = \begin{pmatrix} 0 & -\beta S_0 \\ 0 & \beta S_0 - \gamma \end{pmatrix}$
  - All equilibria are *non-hyperbolic*.

The SIR model: Analysis (equilibria *etc.*)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- **Equilibria:** $(S, I) = (S_0, 0)$ for any $S_0 \in [0, 1]$
  - Continuum of equilibria.
  - Does this make sense?
  - Biological meaning of equilibria?

- **Linearization:**
  - $DF_{(S,I)} = \begin{pmatrix} -\beta I & -\beta S \\ \beta I & \beta S - \gamma \end{pmatrix}$
  - $DF_{(S_0,0)} = \begin{pmatrix} 0 & -\beta S_0 \\ 0 & \beta S_0 - \gamma \end{pmatrix}$
  - All equilibria are *non-hyperbolic*.
- **Periodic orbits:**

The SIR model: Analysis (equilibria *etc.*)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- **Equilibria:** $(S, I) = (S_0, 0)$ for any $S_0 \in [0, 1]$
  - Continuum of equilibria.
  - Does this make sense?
  - Biological meaning of equilibria?

- **Linearization:**
  - $DF_{(S,I)} = \begin{pmatrix} -\beta I & -\beta S \\ \beta I & \beta S - \gamma \end{pmatrix}$
  - $DF_{(S_0,0)} = \begin{pmatrix} 0 & -\beta S_0 \\ 0 & \beta S_0 - \gamma \end{pmatrix}$
  - All equilibria are *non-hyperbolic*.
- **Periodic orbits:**
  - Please do Poll 2 on childsmath.

The SIR model: Analysis (equilibria *etc.*)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- **Equilibria:** $(S, I) = (S_0, 0)$ for any $S_0 \in [0, 1]$
  - Continuum of equilibria.
  - Does this make sense?
  - Biological meaning of equilibria?

- **Linearization:**
  - $DF_{(S,I)} = \begin{pmatrix} -\beta I & -\beta S \\ \beta I & \beta S - \gamma \end{pmatrix}$
  - $DF_{(S_0,0)} = \begin{pmatrix} 0 & -\beta S_0 \\ 0 & \beta S_0 - \gamma \end{pmatrix}$
  - All equilibria are *non-hyperbolic*.
- **Periodic orbits:**
  - Please do Poll 2 on childsmath.
  - $(S + I)' = -\gamma I$ $\Longrightarrow$

The SIR model: Analysis (equilibria *etc.*)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- **Equilibria:**
  $(S, I) = (S_0, 0)$ for any $S_0 \in [0, 1]$
  - Continuum of equilibria.
  - Does this make sense?
  - Biological meaning of equilibria?

- **Linearization:**
  - $DF_{(S,I)} = \begin{pmatrix} -\beta I & -\beta S \\ \beta I & \beta S - \gamma \end{pmatrix}$
  - $DF_{(S_0,0)} = \begin{pmatrix} 0 & -\beta S_0 \\ 0 & \beta S_0 - \gamma \end{pmatrix}$
  - All equilibria are *non-hyperbolic*.

- **Periodic orbits:**
  - Please do Poll 2 on `childsmath`.
  - $(S + I)' = -\gamma I$
    $\implies$ no periodic orbits. Why?

The SIR model: Analysis (equilibria *etc.*)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- **Equilibria:**
  $(S, I) = (S_0, 0)$ for any $S_0 \in [0, 1]$
  - Continuum of equilibria.
  - Does this make sense?
  - Biological meaning of equilibria?

- **Linearization:**
  - $DF_{(S,I)} = \begin{pmatrix} -\beta I & -\beta S \\ \beta I & \beta S - \gamma \end{pmatrix}$
  - $DF_{(S_0,0)} = \begin{pmatrix} 0 & -\beta S_0 \\ 0 & \beta S_0 - \gamma \end{pmatrix}$
  - All equilibria are *non-hyperbolic*.

- **Periodic orbits:**
  - Please do Poll 2 on childsmath.
  - $(S + I)' = -\gamma I$
    $\implies$ no periodic orbits. Why?
    - If $I(0) = 0$ then

The SIR model: Analysis (equilibria *etc.*)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- **Equilibria:** $(S, I) = (S_0, 0)$ for any $S_0 \in [0, 1]$
  - Continuum of equilibria.
  - Does this make sense?
  - Biological meaning of equilibria?

- **Linearization:**
  - $DF_{(S,I)} = \begin{pmatrix} -\beta I & -\beta S \\ \beta I & \beta S - \gamma \end{pmatrix}$
  - $DF_{(S_0,0)} = \begin{pmatrix} 0 & -\beta S_0 \\ 0 & \beta S_0 - \gamma \end{pmatrix}$
  - All equilibria are *non-hyperbolic*.

- **Periodic orbits:**
  - Please do Poll 2 on childsmath.
  - $(S + I)' = -\gamma I$ $\implies$ no periodic orbits. Why?
    - If $I(0) = 0$ then equilibrium.

The SIR model: Analysis (equilibria *etc.*)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- **Equilibria:**
  $(S, I) = (S_0, 0)$ for any $S_0 \in [0, 1]$
  - Continuum of equilibria.
  - Does this make sense?
  - Biological meaning of equilibria?

- **Linearization:**
  - $DF_{(S,I)} = \begin{pmatrix} -\beta I & -\beta S \\ \beta I & \beta S - \gamma \end{pmatrix}$
  - $DF_{(S_0,0)} = \begin{pmatrix} 0 & -\beta S_0 \\ 0 & \beta S_0 - \gamma \end{pmatrix}$
  - All equilibria are *non-hyperbolic*.

- **Periodic orbits:**
  - Please do Poll 2 on `childsmath`.
  - $(S + I)' = -\gamma I$
    $\implies$ no periodic orbits. Why?
    - If $I(0) = 0$ then equilibrium.
    - If $I(0) > 0$ then

The SIR model: Analysis (equilibria *etc.*)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- **Equilibria:**
  $(S, I) = (S_0, 0)$ for any $S_0 \in [0, 1]$
  - Continuum of equilibria.
  - Does this make sense?
  - Biological meaning of equilibria?

- **Linearization:**
  - $DF_{(S,I)} = \begin{pmatrix} -\beta I & -\beta S \\ \beta I & \beta S - \gamma \end{pmatrix}$
  - $DF_{(S_0,0)} = \begin{pmatrix} 0 & -\beta S_0 \\ 0 & \beta S_0 - \gamma \end{pmatrix}$
  - All equilibria are *non-hyperbolic*.

- **Periodic orbits:**
  - Please do Poll 2 on childsmath.
  - $(S + I)' = -\gamma I$
    $\implies$ no periodic orbits. Why?
    - If $I(0) = 0$ then equilibrium.
    - If $I(0) > 0$ then $(S + I)' < 0$,

The SIR model: Analysis (equilibria *etc.*)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- **Equilibria:** $(S, I) = (S_0, 0)$ for any $S_0 \in [0, 1]$
  - Continuum of equilibria.
  - Does this make sense?
  - Biological meaning of equilibria?

- **Linearization:**
  - $DF_{(S,I)} = \begin{pmatrix} -\beta I & -\beta S \\ \beta I & \beta S - \gamma \end{pmatrix}$
  - $DF_{(S_0,0)} = \begin{pmatrix} 0 & -\beta S_0 \\ 0 & \beta S_0 - \gamma \end{pmatrix}$
  - All equilibria are *non-hyperbolic*.
- **Periodic orbits:**
  - Please do Poll 2 on childsmath.
  - $(S + I)' = -\gamma I$
    $\implies$ no periodic orbits. Why?
    - If $I(0) = 0$ then equilibrium.
    - If $I(0) > 0$ then $(S + I)' < 0$, so cannot increase back to initial state.

The SIR model: Analysis (equilibria *etc.*)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- **Equilibria:** $(S, I) = (S_0, 0)$ for any $S_0 \in [0, 1]$
  - Continuum of equilibria.
  - Does this make sense?
  - Biological meaning of equilibria?

- **Linearization:**
  - $DF_{(S,I)} = \begin{pmatrix} -\beta I & -\beta S \\ \beta I & \beta S - \gamma \end{pmatrix}$
  - $DF_{(S_0,0)} = \begin{pmatrix} 0 & -\beta S_0 \\ 0 & \beta S_0 - \gamma \end{pmatrix}$
  - All equilibria are *non-hyperbolic*.
- **Periodic orbits:**
  - Please do Poll 2 on childsmath.
  - $(S + I)' = -\gamma I$
    $\implies$ no periodic orbits. Why?
    - If $I(0) = 0$ then equilibrium.
    - If $I(0) > 0$ then $(S + I)' < 0$, so cannot increase back to initial state.
  - Also follows from Index Theorem

The SIR model: Analysis (equilibria *etc.*)

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

- **Equilibria:** $(S, I) = (S_0, 0)$ for any $S_0 \in [0, 1]$
  - Continuum of equilibria.
  - Does this make sense?
  - Biological meaning of equilibria?

- **Linearization:**
  - $DF_{(S,I)} = \begin{pmatrix} -\beta I & -\beta S \\ \beta I & \beta S - \gamma \end{pmatrix}$
  - $DF_{(S_0,0)} = \begin{pmatrix} 0 & -\beta S_0 \\ 0 & \beta S_0 - \gamma \end{pmatrix}$
  - All equilibria are *non-hyperbolic*.
- **Periodic orbits:**
  - Please do Poll 2 on `childsmath`.
  - $(S + I)' = -\gamma I$
    $\implies$ no periodic orbits. Why?
    - If $I(0) = 0$ then equilibrium.
    - If $I(0) > 0$ then $(S + I)' < 0$, so cannot increase back to initial state.
  - Also follows from Index Theorem (cannot enclose any equilibria).

The SIR model: Analysis

Nullclines:

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

The SIR model: Analysis

Nullclines:

- $S' = 0 \implies$

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

The SIR model: Analysis

Nullclines:

- $S' = 0 \implies S = 0$ or $I = 0$

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

The SIR model: Analysis

Nullclines:

- $S' = 0 \implies S = 0$ or $I = 0$
  - S nullclines: both coordinate axes

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

The SIR model: Analysis

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Nullclines:

- $S' = 0 \implies S = 0$ or $I = 0$
  - S nullclines: both coordinate axes
- $I' = 0 \implies$

The SIR model: Analysis

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Nullclines:

- $S' = 0 \implies S = 0$ or $I = 0$
  - S nullclines: both coordinate axes
- $I' = 0 \implies I = 0$ or $S = \gamma/\beta$

The SIR model: Analysis

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Nullclines:

- $S' = 0 \implies S = 0$ or $I = 0$
  - S nullclines: both coordinate axes
- $I' = 0 \implies I = 0$ or $S = \gamma/\beta$
  - I nullclines: S axis and vertical line at $S = 1/\mathcal{R}_0$

The SIR model: Analysis

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Nullclines:

- $S' = 0 \implies S = 0$ or $I = 0$
  - S nullclines: both coordinate axes
- $I' = 0 \implies I = 0$ or $S = \gamma/\beta$
  - I nullclines: S axis and vertical line at $S = 1/\mathcal{R}_0$
  - Is the I nullcline at $S = 1/\mathcal{R}_0$ always relevant?

The SIR model: Analysis

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Nullclines:

- $S' = 0 \implies S = 0$ or $I = 0$
  - S nullclines: both coordinate axes
- $I' = 0 \implies I = 0$ or $S = \gamma/\beta$
  - I nullclines: S axis and vertical line at $S = 1/\mathcal{R}_0$
  - Is the I nullcline at $S = 1/\mathcal{R}_0$ always relevant?
    - If, and only if, $\mathcal{R}_0 > 1$.

The SIR model: Analysis

$$\frac{dS}{dt} = -\beta SI$$
$$\frac{dI}{dt} = \beta SI - \gamma I$$

Nullclines:

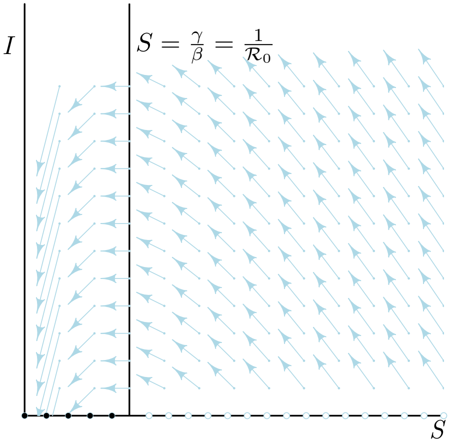
- $S' = 0 \implies S = 0$ or $I = 0$
  - S nullclines: both coordinate axes
- $I' = 0 \implies I = 0$ or $S = \gamma/\beta$
  - I nullclines: S axis and vertical line at $S = 1/\mathcal{R}_0$
  - Is the I nullcline at $S = 1/\mathcal{R}_0$ always relevant?
    - If, and only if, $\mathcal{R}_0 > 1$.
    - If $\mathcal{R}_0 < 1$ then $S = 1/\mathcal{R}_0$ is outside the biologically relevant region of the (S, I) phase plane.

The SIR model: Analysis

Nullclines and Direction Field ($\mathcal{R}_0 = 4$):

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$



The SIR model: Analysis

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Phase Portrait:

The SIR model: Analysis

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Phase Portrait:

- We cannot find solutions $S(t)$ and $I(t)$ for this system.

The SIR model: Analysis

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Phase Portrait:

- We cannot find solutions $S(t)$ and $I(t)$ for this system.
- We *can* find exact analytical solution for the phase portrait!

The SIR model: Analysis

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Phase Portrait:

- We cannot find solutions $S(t)$ and $I(t)$ for this system.
- We *can* find exact analytical solution for the phase portrait!
- *i.e.*, we can find an expression $I(S)$ for solution curves in the (S, I) phase plane.

The SIR model: Analysis

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Phase Portrait:

- We cannot find solutions $S(t)$ and $I(t)$ for this system.
- We *can* find exact analytical solution for the phase portrait!

- *i.e.*, we can find an expression $I(S)$ for solution curves in the (S, I) phase plane.
- Slope of $I(S)$ depends only on S:

$$\frac{dI}{dS} =$$

The SIR model: Analysis

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Phase Portrait:

- We cannot find solutions $S(t)$ and $I(t)$ for this system.
- We *can* find exact analytical solution for the phase portrait!

- *i.e.*, we can find an expression $I(S)$ for solution curves in the (S, I) phase plane.
- Slope of $I(S)$ depends only on S:

$$\frac{dI}{dS} = \frac{dI/dt}{dS/dt} =$$

The SIR model: Analysis

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Phase Portrait:

- We cannot find solutions $S(t)$ and $I(t)$ for this system.
- We *can* find exact analytical solution for the phase portrait!

- *i.e.*, we can find an expression $I(S)$ for solution curves in the (S, I) phase plane.
- Slope of $I(S)$ depends only on S:

$$\frac{dI}{dS} = \frac{dI/dt}{dS/dt} = -1 + \frac{1}{\mathcal{R}_0 S} \qquad (*)$$

The SIR model: Analysis

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Phase Portrait:

- We cannot find solutions $S(t)$ and $I(t)$ for this system.
- We *can* find exact analytical solution for the phase portrait!

- *i.e.*, we can find an expression $I(S)$ for solution curves in the (S, I) phase plane.
- Slope of $I(S)$ depends only on S:

$$\frac{dI}{dS} = \frac{dI/dt}{dS/dt} = -1 + \frac{1}{\mathcal{R}_0 S} \qquad (*)$$

- *Note:* Slope is flat for $S = 1/\mathcal{R}_0$, so max or min of $I(S)$ occurs on I nullcline if $\mathcal{R}_0 > 1$

The SIR model: Analysis

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Phase Portrait:

- We cannot find solutions $S(t)$ and $I(t)$ for this system.
- We *can* find exact analytical solution for the phase portrait!

- *i.e.*, we can find an expression $I(S)$ for solution curves in the (S, I) phase plane.
- Slope of $I(S)$ depends only on S:

$$\frac{dI}{dS} = \frac{dI/dt}{dS/dt} = -1 + \frac{1}{\mathcal{R}_0 S} \qquad (*)$$

- *Note:* Slope is flat for $S = 1/\mathcal{R}_0$, so max or min of $I(S)$ occurs on I nullcline if $\mathcal{R}_0 > 1$
- Easy to integrate $(*)$: $\int_{I_0}^{I} dI = \int_{S_0}^{S} \left(-1 + \frac{1}{\mathcal{R}_0 S}\right) dS$

The SIR model: Analysis

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Phase Portrait:

- We cannot find solutions $S(t)$ and $I(t)$ for this system.
- We *can* find exact analytical solution for the phase portrait!

- *i.e.*, we can find an expression $I(S)$ for solution curves in the (S, I) phase plane.
- Slope of $I(S)$ depends only on S:

$$\frac{dI}{dS} = \frac{dI/dt}{dS/dt} = -1 + \frac{1}{\mathcal{R}_0 S} \qquad (*)$$

- *Note:* Slope is flat for $S = 1/\mathcal{R}_0$, so max or min of $I(S)$ occurs on I nullcline if $\mathcal{R}_0 > 1$
- Easy to integrate $(*)$: $\int_{I_0}^{I} dI = \int_{S_0}^{S} \left(-1 + \frac{1}{\mathcal{R}_0 S}\right) dS$
- $I - I_0 = -(S - S_0) + \frac{1}{\mathcal{R}_0} \log\left(S/S_0\right)$

The SIR model: Analysis

Model Equations:

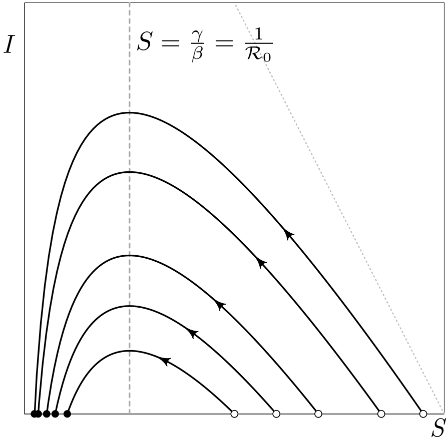
$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Solution Curves in Phase Plane:

$$I + S - (I_0 + S_0) = \frac{1}{\mathcal{R}_0} \log\left(S/S_0\right)$$

Phase Portrait ($\mathcal{R}_0 = 4$):



The SIR model: Analysis

Model Equations:

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Solution Curves in Phase Plane:

$$I + S - (I_0 + S_0) = \frac{1}{\mathcal{R}_0} \log (S/S_0)$$

Final Size of Epidemic:

The SIR model: Analysis

Model Equations:

$$\frac{dS}{dt} = -\beta S I$$

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

Solution Curves in Phase Plane:

$$I + S - (I_0 + S_0) = \frac{1}{\mathcal{R}_0} \log (S/S_0)$$

Final Size of Epidemic:

- As $t \to \infty$ we have $(I_\infty + S_\infty) - (I_0 + S_0) = \frac{1}{\mathcal{R}_0} \log S_\infty / S_0$

The SIR model: Analysis

Model Equations:

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Solution Curves in Phase Plane:

$$I + S - (I_0 + S_0) = \frac{1}{\mathcal{R}_0} \log\left(S/S_0\right)$$

Final Size of Epidemic:

- As $t \to \infty$ we have $(I_\infty + S_\infty) - (I_0 + S_0) = \frac{1}{\mathcal{R}_0} \log S_\infty / S_0$
- But for a newly invading pathogen:

The SIR model: Analysis

Model Equations:

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Solution Curves in Phase Plane:

$$I + S - (I_0 + S_0) = \frac{1}{\mathcal{R}_0} \log(S/S_0)$$

Final Size of Epidemic:

- As $t \to \infty$ we have $(I_\infty + S_\infty) - (I_0 + S_0) = \frac{1}{\mathcal{R}_0} \log S_\infty / S_0$
- But for a newly invading pathogen: $S_0 \simeq 1$, $I_0 \simeq 0$, $I_\infty = 0$

The SIR model: Analysis

Model Equations:

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Solution Curves in Phase Plane:

$$I + S - (I_0 + S_0) = \frac{1}{\mathcal{R}_0} \log (S/S_0)$$

Final Size of Epidemic:

- As $t \to \infty$ we have $(I_\infty + S_\infty) - (I_0 + S_0) = \frac{1}{\mathcal{R}_0} \log S_\infty / S_0$
- But for a newly invading pathogen: $S_0 \simeq 1,\ I_0 \simeq 0,\ I_\infty = 0$
- In the limit $I_0 \to 0$, we have

The SIR model: Analysis

Model Equations:

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Solution Curves in Phase Plane:

$$I + S - (I_0 + S_0) = \frac{1}{\mathcal{R}_0} \log (S/S_0)$$

Final Size of Epidemic:

- As $t \to \infty$ we have $(I_\infty + S_\infty) - (I_0 + S_0) = \frac{1}{\mathcal{R}_0} \log S_\infty / S_0$
- But for a newly invading pathogen: $S_0 \simeq 1$, $I_0 \simeq 0$, $I_\infty = 0$
- In the limit $I_0 \to 0$, we have $(S_\infty - 1) = \frac{1}{\mathcal{R}_0} \log S_\infty$

The SIR model: Analysis

Model Equations:

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Solution Curves in Phase Plane:

$$I + S - (I_0 + S_0) = \frac{1}{\mathcal{R}_0} \log (S/S_0)$$

Final Size of Epidemic:

- As $t \to \infty$ we have $(I_\infty + S_\infty) - (I_0 + S_0) = \frac{1}{\mathcal{R}_0} \log S_\infty / S_0$
- But for a newly invading pathogen: $S_0 \simeq 1,\ I_0 \simeq 0,\ I_\infty = 0$
- In the limit $I_0 \to 0$, we have $(S_\infty - 1) = \frac{1}{\mathcal{R}_0} \log S_\infty$
- Define "Final Size" $Z = 1 - S_\infty$

The SIR model: Analysis

Model Equations:

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Solution Curves in Phase Plane:

$$I + S - (I_0 + S_0)$$
$$= \frac{1}{\mathcal{R}_0} \log (S/S_0)$$

Final Size of Epidemic:

- As $t \to \infty$ we have $(I_\infty + S_\infty) - (I_0 + S_0) = \frac{1}{\mathcal{R}_0} \log S_\infty / S_0$
- But for a newly invading pathogen: $S_0 \simeq 1,\ I_0 \simeq 0,\ I_\infty = 0$
- In the limit $I_0 \to 0$, we have $(S_\infty - 1) = \frac{1}{\mathcal{R}_0} \log S_\infty$
- Define "Final Size" $Z = 1 - S_\infty$
- $\therefore -Z = \frac{1}{\mathcal{R}_0} \log (1 - Z)$

The SIR model: Analysis

Model Equations:

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Solution Curves in Phase Plane:

$$I + S - (I_0 + S_0) = \frac{1}{\mathcal{R}_0} \log\left(S/S_0\right)$$

Final Size of Epidemic:

- As $t \to \infty$ we have $(I_\infty + S_\infty) - (I_0 + S_0) = \frac{1}{\mathcal{R}_0} \log S_\infty / S_0$
- But for a newly invading pathogen: $S_0 \simeq 1,\ I_0 \simeq 0,\ I_\infty = 0$
- In the limit $I_0 \to 0$, we have $(S_\infty - 1) = \frac{1}{\mathcal{R}_0} \log S_\infty$
- Define "Final Size" $Z = 1 - S_\infty$
- $\therefore\ -Z = \frac{1}{\mathcal{R}_0} \log\left(1 - Z\right)$, *i.e.*,

$$Z = 1 - e^{-\mathcal{R}_0 Z}$$

The SIR model: Analysis

Model Equations:

$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I$$

Solution Curves in Phase Plane:

$$I + S - (I_0 + S_0) = \frac{1}{\mathcal{R}_0} \log\left(S/S_0\right)$$

Final Size of Epidemic:

- As $t \to \infty$ we have $(I_\infty + S_\infty) - (I_0 + S_0) = \frac{1}{\mathcal{R}_0} \log S_\infty / S_0$
- But for a newly invading pathogen: $S_0 \simeq 1$, $I_0 \simeq 0$, $I_\infty = 0$
- In the limit $I_0 \to 0$, we have $(S_\infty - 1) = \frac{1}{\mathcal{R}_0} \log S_\infty$
- Define "Final Size" $Z = 1 - S_\infty$
- $\therefore -Z = \frac{1}{\mathcal{R}_0} \log\left(1 - Z\right)$, *i.e.*,

$$Z = 1 - e^{-\mathcal{R}_0 Z}$$

- This is a famous formula, derived by Kermack and McKendrick in 1927.

The SIR model: Analysis

Final Size Formula:

$$Z = 1 - e^{-\mathcal{R}_0 Z}$$

The SIR model: Analysis

Final Size Formula:

$$Z = 1 - e^{-\mathcal{R}_0 Z}$$

- Final size is determined entirely by $\mathcal{R}_0$

The SIR model: Analysis

Final Size Formula:

$$Z = 1 - e^{-\mathcal{R}_0 Z}$$

- Final size is determined entirely by $\mathcal{R}_0$
- Final size is never the whole population ($Z < 1$)

The SIR model: Analysis

Final Size Formula:

$$Z = 1 - e^{-\mathcal{R}_0 Z}$$

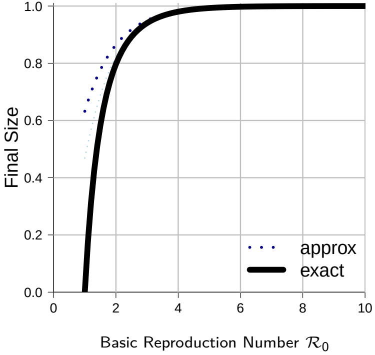
- Final size is determined entirely by $\mathcal{R}_0$
- Final size is never the whole population ($Z < 1$)
- Formula is valid for much more realistic models (Ma & Earn, 2006)

The SIR model: Analysis

Final Size Formula:

$$Z = 1 - e^{-\mathcal{R}_0 Z}$$

- Final size is determined entirely by $\mathcal{R}_0$
- Final size is never the whole population ($Z < 1$)
- Formula is valid for much more realistic models (Ma & Earn, 2006)

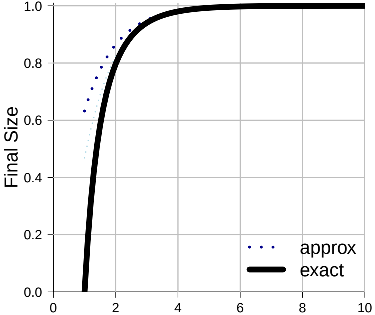


The SIR model: Analysis

Final Size Formula:

$$Z = 1 - e^{-\mathcal{R}_0 Z}$$

- Final size is determined entirely by $\mathcal{R}_0$
- Final size is never the whole population ($Z < 1$)
- Formula is valid for much more realistic models (Ma & Earn, 2006)



Basic Reproduction Number $\mathcal{R}_0$

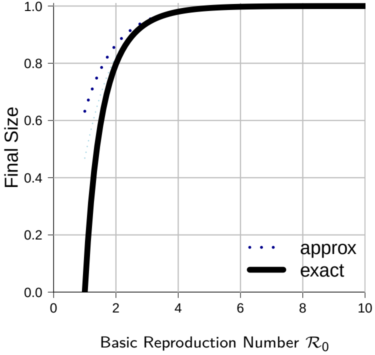
- For 1918 flu:

The SIR model: Analysis

Final Size Formula:

$$Z = 1 - e^{-\mathcal{R}_0 Z}$$

- Final size is determined entirely by $\mathcal{R}_0$
- Final size is never the whole population ($Z < 1$)
- Formula is valid for much more realistic models (Ma & Earn, 2006)



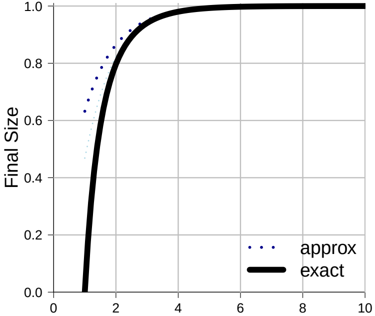
- For 1918 flu: $1.5 \lesssim \mathcal{R}_0 \lesssim 2$

The SIR model: Analysis

Final Size Formula:

$$Z = 1 - e^{-\mathcal{R}_0 Z}$$

- Final size is determined entirely by $\mathcal{R}_0$
- Final size is never the whole population ($Z < 1$)
- Formula is valid for much more realistic models (Ma & Earn, 2006)



Basic Reproduction Number $\mathcal{R}_0$

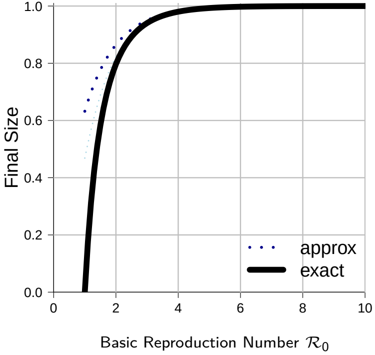
- For 1918 flu: $1.5 \lesssim \mathcal{R}_0 \lesssim 2$
- Proportion of world population infected?

The SIR model: Analysis

Final Size Formula:

$$Z = 1 - e^{-\mathcal{R}_0 Z}$$

- Final size is determined entirely by $\mathcal{R}_0$
- Final size is never the whole population ($Z < 1$)
- Formula is valid for much more realistic models (Ma & Earn, 2006)



- For 1918 flu: $1.5 \lesssim \mathcal{R}_0 \lesssim 2$
- Proportion of world population infected?
- $\sim$ 60–80%

From Final Size to Reproduction Number

From Final Size to Reproduction Number

- The final size relation allows us to estimate the proportion of the population that will be infected *given* an estimate of $\mathcal{R}_0$.

From Final Size to Reproduction Number

- The final size relation allows us to estimate the proportion of the population that will be infected *given* an estimate of $\mathcal{R}_0$.
- But we can turn it around: if we know the final size Z then we can easily estimate $\mathcal{R}_0$:

$$Z = 1 - e^{-\mathcal{R}_0 Z} \quad \Longrightarrow \quad \mathcal{R}_0 = -\frac{1}{Z}\log\left(1 - Z\right)$$

From Final Size to Reproduction Number

- The final size relation allows us to estimate the proportion of the population that will be infected *given* an estimate of $\mathcal{R}_0$.
- But we can turn it around: if we know the final size Z then we can easily estimate $\mathcal{R}_0$:

$$Z = 1 - e^{-\mathcal{R}_0 Z} \quad \Longrightarrow \quad \mathcal{R}_0 = -\frac{1}{Z}\log(1 - Z)$$

- This is useful *post-hoc* only (*after* an epidemic).

The SIR model: Non-dimensionalization

The SIR model: Non-dimensionalization

- Often helpful to use dimensionless parameters.

The SIR model: Non-dimensionalization

- Often helpful to use dimensionless parameters.
- How do we identify "the right" dimensionless parameters?

The SIR model: Non-dimensionalization

- Often helpful to use dimensionless parameters.
- How do we identify "the right" dimensionless parameters?
- $\frac{\beta}{\gamma}$? $\frac{\gamma}{\beta}$? $\frac{\beta}{\beta+\gamma}$? $\frac{\beta^2}{\beta^2+\gamma^2}$?

The SIR model: Non-dimensionalization

- Often helpful to use dimensionless parameters.
- How do we identify "the right" dimensionless parameters?
- $\frac{\beta}{\gamma}$? $\frac{\gamma}{\beta}$? $\frac{\beta}{\beta+\gamma}$? $\frac{\beta^2}{\beta^2+\gamma^2}$?
- We choose β/γ because it has a natural interpretation.

The SIR model: Non-dimensionalization

- Often helpful to use dimensionless parameters.
- How do we identify "the right" dimensionless parameters?
- $\frac{\beta}{\gamma}$? $\frac{\gamma}{\beta}$? $\frac{\beta}{\beta+\gamma}$? $\frac{\beta^2}{\beta^2+\gamma^2}$?
- We choose β/γ because it has a natural interpretation.
- But we are still left with γ as a second parameter.

The SIR model: Non-dimensionalization

- Often helpful to use dimensionless parameters.
- How do we identify "the right" dimensionless parameters?
- $\frac{\beta}{\gamma}$? $\frac{\gamma}{\beta}$? $\frac{\beta}{\beta+\gamma}$? $\frac{\beta^2}{\beta^2+\gamma^2}$?
- We choose β/γ because it has a natural interpretation.
- But we are still left with γ as a second parameter.
- Can we simplify the model somehow?

The SIR model: Non-dimensionalization

- Often helpful to use dimensionless parameters.
- How do we identify "the right" dimensionless parameters?
- $\frac{\beta}{\gamma}$? $\frac{\gamma}{\beta}$? $\frac{\beta}{\beta+\gamma}$? $\frac{\beta^2}{\beta^2+\gamma^2}$?
- We choose β/γ because it has a natural interpretation.
- But we are still left with γ as a second parameter.
- Can we simplify the model somehow?
- γ defines a time scale ($1/\gamma$ is the mean infectious period).

The SIR model: Non-dimensionalization

- Often helpful to use dimensionless parameters.
- How do we identify "the right" dimensionless parameters?
- $\frac{\beta}{\gamma}$? $\frac{\gamma}{\beta}$? $\frac{\beta}{\beta+\gamma}$? $\frac{\beta^2}{\beta^2+\gamma^2}$?
- We choose β/γ because it has a natural interpretation.
- But we are still left with γ as a second parameter.
- Can we simplify the model somehow?
- γ defines a time scale ($1/\gamma$ is the mean infectious period).
- If time unit is mean infectious period, then $\gamma =$

The SIR model: Non-dimensionalization

- Often helpful to use dimensionless parameters.
- How do we identify "the right" dimensionless parameters?
- $\frac{\beta}{\gamma}$? $\frac{\gamma}{\beta}$? $\frac{\beta}{\beta+\gamma}$? $\frac{\beta^2}{\beta^2+\gamma^2}$?
- We choose β/γ because it has a natural interpretation.
- But we are still left with γ as a second parameter.
- Can we simplify the model somehow?
- γ defines a time scale ($1/\gamma$ is the mean infectious period).
- If time unit is mean infectious period, then $\gamma = 1$.

The SIR model: Non-dimensionalization

- Often helpful to use dimensionless parameters.
- How do we identify "the right" dimensionless parameters?
- $\frac{\beta}{\gamma}$? $\quad \frac{\gamma}{\beta}$? $\quad \frac{\beta}{\beta+\gamma}$? $\quad \frac{\beta^2}{\beta^2+\gamma^2}$?
- We choose β/γ because it has a natural interpretation.
- But we are still left with γ as a second parameter.
- Can we simplify the model somehow?
- γ defines a time scale ($1/\gamma$ is the mean infectious period).
- If time unit is mean infectious period, then $\gamma = 1$.
- So in these "natural" time units, the SIR model is

$$\frac{dS}{dt} = -\mathcal{R}_0 SI, \quad \frac{dI}{dt} = \mathcal{R}_0 SI - I$$

The SIR model: Non-dimensionalization

- Often helpful to use dimensionless parameters.
- How do we identify "the right" dimensionless parameters?
- $\frac{\beta}{\gamma}$? $\quad \frac{\gamma}{\beta}$? $\quad \frac{\beta}{\beta+\gamma}$? $\quad \frac{\beta^2}{\beta^2+\gamma^2}$?
- We choose β/γ because it has a natural interpretation.
- But we are still left with γ as a second parameter.
- Can we simplify the model somehow?
- γ defines a time scale ($1/\gamma$ is the mean infectious period).
- If time unit is mean infectious period, then $\gamma = 1$.
- So in these "natural" time units, the SIR model is

$$\frac{dS}{dt} = -\mathcal{R}_0 SI, \quad \frac{dI}{dt} = \mathcal{R}_0 SI - I$$

- There is really only one parameter in the model. The other is just a time scale and does not affect the *qualitative* dynamics.

The SIR model: Results so far

Mathematical Results:

The SIR model: Results so far

Mathematical Results:

- Model is biologically well-posed

The SIR model: Results so far

Mathematical Results:

- Model is biologically well-posed
  - $0 \leq S(0) + I(0) \leq 1 \quad \Longrightarrow \quad 0 \leq S(t) + I(t) \leq 1 \quad \forall t > 0$

The SIR model: Results so far

Mathematical Results:

- Model is biologically well-posed
  - $0 \leq S(0) + I(0) \leq 1 \quad \Longrightarrow \quad 0 \leq S(t) + I(t) \leq 1 \quad \forall t > 0$
- No periodic orbits.

The SIR model: Results so far

Mathematical Results:

- Model is biologically well-posed
  - $0 \leq S(0) + I(0) \leq 1 \implies 0 \leq S(t) + I(t) \leq 1 \quad \forall t > 0$
- No periodic orbits.
- Continuum of equilibria.

The SIR model: Results so far

Mathematical Results:

- Model is biologically well-posed
  - $0 \leq S(0) + I(0) \leq 1 \implies 0 \leq S(t) + I(t) \leq 1 \quad \forall t > 0$
- No periodic orbits.
- Continuum of equilibria.
- Stability of equilibria:

The SIR model: Results so far

Mathematical Results:

- Model is biologically well-posed
  - $0 \leq S(0) + I(0) \leq 1 \implies 0 \leq S(t) + I(t) \leq 1 \quad \forall t > 0$
- No periodic orbits.
- Continuum of equilibria.
- Stability of equilibria:
  - Linearization useless (all equilibria non-hyperbolic).

The SIR model: Results so far

Mathematical Results:

- Model is biologically well-posed
  - $0 \leq S(0) + I(0) \leq 1 \implies 0 \leq S(t) + I(t) \leq 1 \quad \forall t > 0$
- No periodic orbits.
- Continuum of equilibria.
- Stability of equilibria:
  - Linearization useless (all equilibria non-hyperbolic).
  - Further analysis necessary.

The SIR model: Results so far

Mathematical Results:

- Model is biologically well-posed
  - $0 \leq S(0) + I(0) \leq 1 \implies 0 \leq S(t) + I(t) \leq 1 \quad \forall t > 0$
- No periodic orbits.
- Continuum of equilibria.
- Stability of equilibria:
  - Linearization useless (all equilibria non-hyperbolic).
  - Further analysis necessary.
- Exact solution for phase portrait:
  $$I(S) = I_0 + (S_0 - S) + \frac{1}{\mathcal{R}_0} \log\left(S/S_0\right)$$

The SIR model: Results so far

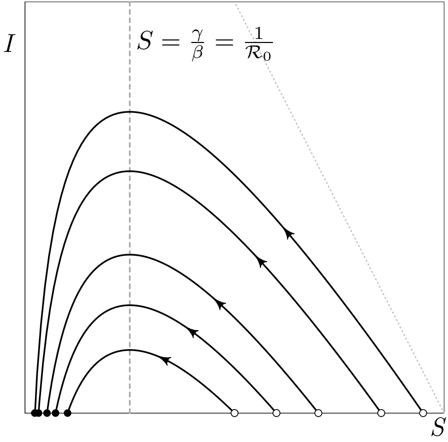
Mathematical Results:

- Model is biologically well-posed
  - $0 \leq S(0) + I(0) \leq 1 \implies 0 \leq S(t) + I(t) \leq 1 \quad \forall t > 0$
- No periodic orbits.
- Continuum of equilibria.
- Stability of equilibria:
  - Linearization useless (all equilibria non-hyperbolic).
  - Further analysis necessary.
- Exact solution for phase portrait:
  $I(S) = I_0 + (S_0 - S) + \frac{1}{\mathcal{R}_0} \log\left(S/S_0\right)$
- Final size formula: $Z = 1 - e^{-\mathcal{R}_0 Z}$

The SIR model: Stability of equilibria

The SIR model: Stability of equilibria

Phase Portrait ($\mathcal{R}_0 = 4$):

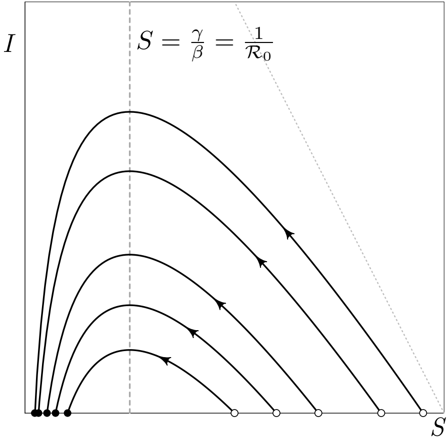


Model Equations:

$$\frac{dS}{dt} = -\beta SI, \qquad \frac{dI}{dt} = \beta SI - \gamma I$$

The SIR model: Stability of equilibria

Phase Portrait ($\mathcal{R}_0 = 4$):



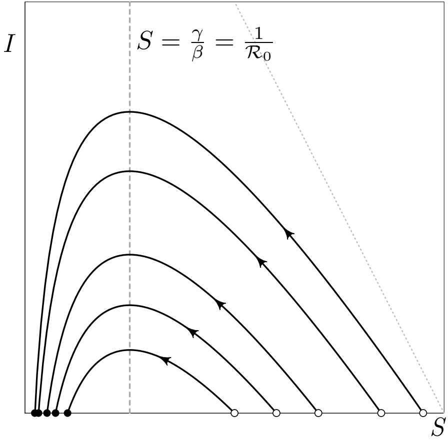
Model Equations:

$$\frac{dS}{dt} = -\beta SI, \qquad \frac{dI}{dt} = \beta SI - \gamma I$$

Which equilibria are:

The SIR model: Stability of equilibria

Phase Portrait ($\mathcal{R}_0 = 4$):



Model Equations:

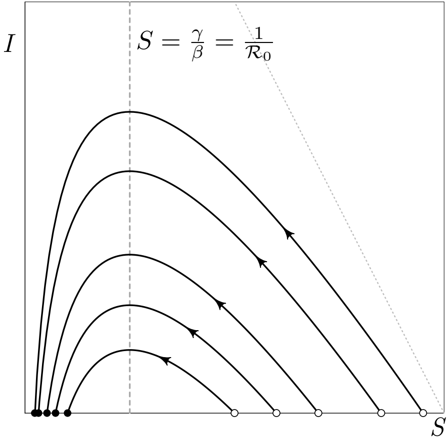
$$\frac{dS}{dt} = -\beta SI, \qquad \frac{dI}{dt} = \beta SI - \gamma I$$

Which equilibria are:

- Unstable?

The SIR model: Stability of equilibria

Phase Portrait ($\mathcal{R}_0 = 4$):



Model Equations:

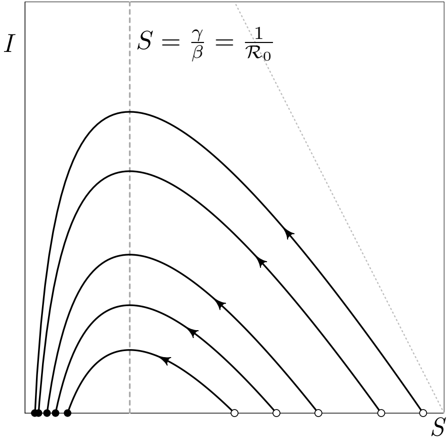
$$\frac{dS}{dt} = -\beta SI, \quad \frac{dI}{dt} = \beta SI - \gamma I$$

Which equilibria are:

- Unstable?
  - $S_0 > 1/\mathcal{R}_0$

The SIR model: Stability of equilibria

Phase Portrait ($\mathcal{R}_0 = 4$):



Model Equations:

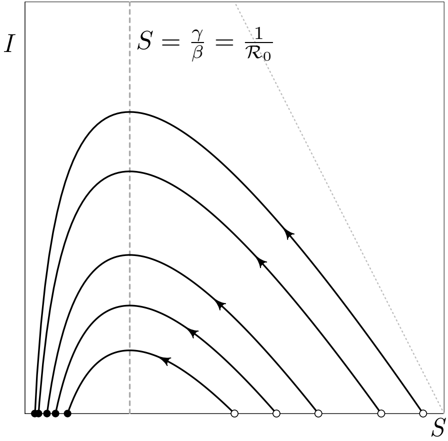
$$\frac{dS}{dt} = -\beta SI, \quad \frac{dI}{dt} = \beta SI - \gamma I$$

Which equilibria are:

- Unstable?
  - $S_0 > 1/\mathcal{R}_0$
- Stable?

The SIR model: Stability of equilibria

Phase Portrait ($\mathcal{R}_0 = 4$):



Model Equations:

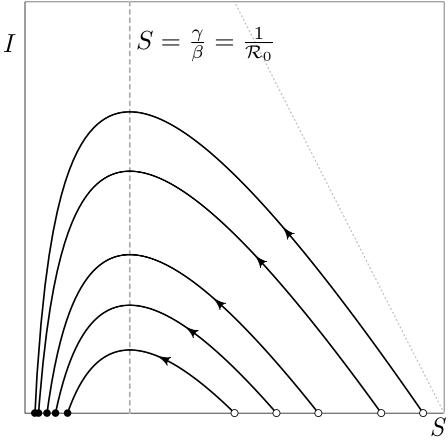
$$\frac{dS}{dt} = -\beta SI, \qquad \frac{dI}{dt} = \beta SI - \gamma I$$

Which equilibria are:

- Unstable?
  - $S_0 > 1/\mathcal{R}_0$
- Stable?
  - $S_0 \le 1/\mathcal{R}_0$

The SIR model: Stability of equilibria

Phase Portrait ($\mathcal{R}_0 = 4$):



Model Equations:

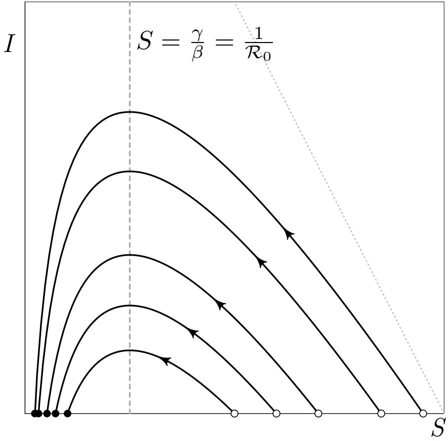
$$\frac{dS}{dt} = -\beta S I, \quad \frac{dI}{dt} = \beta S I - \gamma I$$

Which equilibria are:

- Unstable?
  - $S_0 > 1/\mathcal{R}_0$
- Stable?
  - $S_0 \leq 1/\mathcal{R}_0$
- Asymptotically stable?

The SIR model: Stability of equilibria

Phase Portrait ($\mathcal{R}_0 = 4$):



Model Equations:

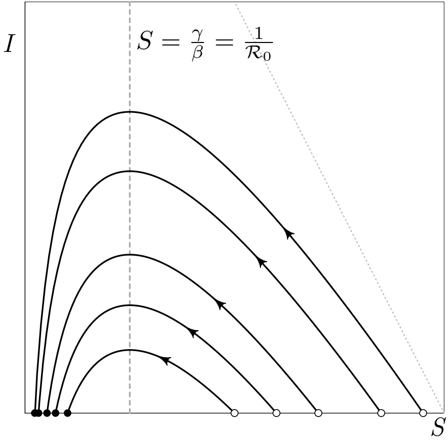
$$\frac{dS}{dt} = -\beta SI, \quad \frac{dI}{dt} = \beta SI - \gamma I$$

Which equilibria are:

- Unstable?
  - $S_0 > 1/\mathcal{R}_0$
- Stable?
  - $S_0 \leq 1/\mathcal{R}_0$
- Asymptotically stable?
  - None!

The SIR model: Stability of equilibria

Phase Portrait ($\mathcal{R}_0 = 4$):



Model Equations:

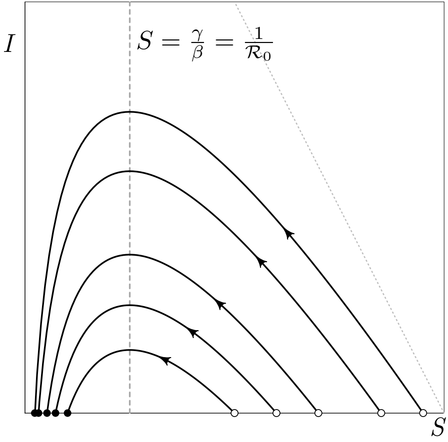
$$\frac{dS}{dt} = -\beta S I, \qquad \frac{dI}{dt} = \beta S I - \gamma I$$

Which equilibria are:

- Unstable?
  - $S_0 > 1/\mathcal{R}_0$
- Stable?
  - $S_0 \le 1/\mathcal{R}_0$
- Asymptotically stable?
  - None!
- How do we prove these facts?

The SIR model: Stability of equilibria

Phase Portrait ($\mathcal{R}_0 = 4$):



Model Equations:

$$\frac{dS}{dt} = -\beta SI, \quad \frac{dI}{dt} = \beta SI - \gamma I$$

Which equilibria are:

- Unstable?
  - $S_0 > 1/\mathcal{R}_0$
- Stable?
  - $S_0 \le 1/\mathcal{R}_0$
- Asymptotically stable?
  - None!
- How do we prove these facts? (**Assignment 1**)

The SIR model: Effects of Control Measures

The SIR model: Effects of Control Measures

- How could the final size of the 1918 pandemic have been reduced?

The SIR model: Effects of Control Measures

- How could the final size of the 1918 pandemic have been reduced?
- Any intervention that reduces $\mathcal{R}_0$ reduces the final size.

The SIR model: Effects of Control Measures

- How could the final size of the 1918 pandemic have been reduced?
- Any intervention that reduces $\mathcal{R}_0$ reduces the final size.
- What could have been done to reduce $\mathcal{R}_0$?

The SIR model: Effects of Control Measures

- How could the final size of the 1918 pandemic have been reduced?
- Any intervention that reduces $\mathcal{R}_0$ reduces the final size.
- What could have been done to reduce $\mathcal{R}_0$?
- Masks?

The SIR model: Effects of Control Measures

- How could the final size of the 1918 pandemic have been reduced?
- Any intervention that reduces $\mathcal{R}_0$ reduces the final size.
- What could have been done to reduce $\mathcal{R}_0$?
- Masks? Quarantine?

The SIR model: Effects of Control Measures

- How could the final size of the 1918 pandemic have been reduced?
- Any intervention that reduces $\mathcal{R}_0$ reduces the final size.
- What could have been done to reduce $\mathcal{R}_0$?
- Masks? Quarantine? Isolation?

The SIR model: Effects of Control Measures

- How could the final size of the 1918 pandemic have been reduced?
- Any intervention that reduces $\mathcal{R}_0$ reduces the final size.
- What could have been done to reduce $\mathcal{R}_0$?
- Masks? Quarantine? Isolation?
- Ideally a vaccine, but no such luck in 1918.

The SIR model: Effects of Control Measures

- How could the final size of the 1918 pandemic have been reduced?
- Any intervention that reduces $\mathcal{R}_0$ reduces the final size.
- What could have been done to reduce $\mathcal{R}_0$?
- Masks? Quarantine? Isolation?
- Ideally a vaccine, but no such luck in 1918.
- Even in 2009, it took months to mass-produce vaccine.

The SIR model: Effects of Control Measures

- How could the final size of the 1918 pandemic have been reduced?
- Any intervention that reduces $\mathcal{R}_0$ reduces the final size.
- What could have been done to reduce $\mathcal{R}_0$?
- Masks? Quarantine? Isolation?
- Ideally a vaccine, but no such luck in 1918.
- Even in 2009, it took months to mass-produce vaccine.
- But suppose there had been a vaccine immediately...

The SIR model: Effects of Control Measures

- How could the final size of the 1918 pandemic have been reduced?
- Any intervention that reduces $\mathcal{R}_0$ reduces the final size.
- What could have been done to reduce $\mathcal{R}_0$?
- Masks? Quarantine? Isolation?
- Ideally a vaccine, but no such luck in 1918.
- Even in 2009, it took months to mass-produce vaccine.
- But suppose there had been a vaccine immediately...
- What proportion (p) of the population do we need to vaccinate to eradicate an infectious disease?

The SIR model: Effects of Control Measures

The SIR model: Effects of Control Measures

Suppose a proportion (p) of the population is vaccinated before an epidemic starts.

The SIR model: Effects of Control Measures

Suppose a proportion (p) of the population is vaccinated before an epidemic starts. Then:

The SIR model: Effects of Control Measures

Suppose a proportion (p) of the population is vaccinated before an epidemic starts. Then:

- At the start of the epidemic, the proportion of the population that is susceptible is $S_0 = 1 - p$.

The SIR model: Effects of Control Measures

Suppose a proportion (p) of the population is vaccinated before an epidemic starts. Then:

- At the start of the epidemic, the proportion of the population that is susceptible is $S_0 = 1 - p$.
- $\therefore$ Initially (at time $t = 0$) the rate of change of prevalence is

$$\left.\frac{dI}{dt}\right|_{t=0} =$$

The SIR model: Effects of Control Measures

Suppose a proportion (p) of the population is vaccinated before an epidemic starts. Then:

- At the start of the epidemic, the proportion of the population that is susceptible is $S_0 = 1 - p$.
- $\therefore$ Initially (at time $t = 0$) the rate of change of prevalence is

$$\left.\frac{dI}{dt}\right|_{t=0} = \left.\Big((\mathcal{R}_0 S - 1)I\Big)\right|_{t=0} =$$

The SIR model: Effects of Control Measures

Suppose a proportion (p) of the population is vaccinated before an epidemic starts. Then:

- At the start of the epidemic, the proportion of the population that is susceptible is $S_0 = 1 - p$.
- $\therefore$ Initially (at time $t = 0$) the rate of change of prevalence is

$$\left.\frac{dI}{dt}\right|_{t=0} = \left.\Big((\mathcal{R}_0 S - 1)\,I\Big)\right|_{t=0} = (\mathcal{R}_0 S_0 - 1)\,I_0$$
$$=$$

The SIR model: Effects of Control Measures

Suppose a proportion (p) of the population is vaccinated before an epidemic starts. Then:

- At the start of the epidemic, the proportion of the population that is susceptible is $S_0 = 1 - p$.
- $\therefore$ Initially (at time $t = 0$) the rate of change of prevalence is

$$\left.\frac{dI}{dt}\right|_{t=0} = \left.\Big((\mathcal{R}_0 S - 1)\, I\Big)\right|_{t=0} = (\mathcal{R}_0 S_0 - 1)\, I_0$$
$$= (\mathcal{R}_0(1 - p) - 1)\, I_0$$

The SIR model: Effects of Control Measures

Suppose a proportion (p) of the population is vaccinated before an epidemic starts. Then:

- At the start of the epidemic, the proportion of the population that is susceptible is $S_0 = 1 - p$.
- $\therefore$ Initially (at time $t = 0$) the rate of change of prevalence is

$$\begin{aligned} \left.\frac{dI}{dt}\right|_{t=0} &= \left.\Big((\mathcal{R}_0 S - 1)\, I\Big)\right|_{t=0} = (\mathcal{R}_0 S_0 - 1)\, I_0 \\ &= (\mathcal{R}_0(1-p) - 1)\, I_0 \quad < 0 \iff \mathcal{R}_0(1-p) < 1 \end{aligned}$$

The SIR model: Effects of Control Measures

Suppose a proportion (p) of the population is vaccinated before an epidemic starts. Then:

- At the start of the epidemic, the proportion of the population that is susceptible is $S_0 = 1 - p$.
- $\therefore$ Initially (at time $t = 0$) the rate of change of prevalence is

$$\left.\frac{dI}{dt}\right|_{t=0} = \left.\Big((\mathcal{R}_0 S - 1)\,I\Big)\right|_{t=0} = (\mathcal{R}_0 S_0 - 1)\,I_0$$
$$= (\mathcal{R}_0(1-p) - 1)\,I_0 \quad < 0 \iff \mathcal{R}_0(1-p) < 1$$

- $\therefore$ An epidemic will be prevented if

$$p >$$

The SIR model: Effects of Control Measures

Suppose a proportion (p) of the population is vaccinated before an epidemic starts. Then:

- At the start of the epidemic, the proportion of the population that is susceptible is $S_0 = 1 - p$.
- $\therefore$ Initially (at time $t = 0$) the rate of change of prevalence is

$$\left.\frac{dI}{dt}\right|_{t=0} = \left.\Big((\mathcal{R}_0 S - 1)\,I\Big)\right|_{t=0} = (\mathcal{R}_0 S_0 - 1)\,I_0$$
$$= (\mathcal{R}_0(1-p) - 1)\,I_0 \quad < 0 \iff \mathcal{R}_0(1-p) < 1$$

- $\therefore$ An epidemic will be prevented if

$$p > p_{\text{crit}} =$$

The SIR model: Effects of Control Measures

Suppose a proportion (p) of the population is vaccinated before an epidemic starts. Then:

- At the start of the epidemic, the proportion of the population that is susceptible is $S_0 = 1 - p$.
- $\therefore$ Initially (at time $t = 0$) the rate of change of prevalence is

$$\left.\frac{dI}{dt}\right|_{t=0} = \left.\Big((\mathcal{R}_0 S - 1)\,I\Big)\right|_{t=0} = (\mathcal{R}_0 S_0 - 1)\,I_0$$
$$= (\mathcal{R}_0(1-p) - 1)\,I_0 \quad < 0 \iff \mathcal{R}_0(1-p) < 1$$

- $\therefore$ An epidemic will be prevented if

$$p > p_{\text{crit}} = 1 - \frac{1}{\mathcal{R}_0}$$

The SIR model: Effects of Control Measures

Suppose a proportion (p) of the population is vaccinated before an epidemic starts. Then:

- At the start of the epidemic, the proportion of the population that is susceptible is $S_0 = 1 - p$.
- $\therefore$ Initially (at time $t = 0$) the rate of change of prevalence is

$$\left.\frac{dI}{dt}\right|_{t=0} = \left.\Big((\mathcal{R}_0 S - 1)\,I\Big)\right|_{t=0} = (\mathcal{R}_0 S_0 - 1)\,I_0$$
$$= (\mathcal{R}_0(1-p) - 1)\,I_0 \quad < 0 \iff \mathcal{R}_0(1-p) < 1$$

- $\therefore$ An epidemic will be prevented if

$$p > p_{\rm crit} = 1 - \frac{1}{\mathcal{R}_0}$$

- $\therefore$ Public Health Agency will ask you to estimate $\mathcal{R}_0$.

The SIR model: Results so far

Biological inferences:

The SIR model: Results so far

Biological inferences:

- $\mathcal{R}_0$ is extremely important to estimate in practice!

The SIR model: Results so far

Biological inferences:

- $\mathcal{R}_0$ is extremely important to estimate in practice!
- Epidemic occurs if and only if $\mathcal{R}_0 > 1$.

The SIR model: Results so far

Biological inferences:

- $\mathcal{R}_0$ is extremely important to estimate in practice!
- Epidemic occurs if and only if $\mathcal{R}_0 > 1$.
- Single epidemic, then disease disappears.

The SIR model: Results so far

Biological inferences:

- $\mathcal{R}_0$ is extremely important to estimate in practice!
- Epidemic occurs if and only if $\mathcal{R}_0 > 1$.
- Single epidemic, then disease disappears.
  - Proof?

The SIR model: Results so far

Biological inferences:

- $\mathcal{R}_0$ is extremely important to estimate in practice!
- Epidemic occurs if and only if $\mathcal{R}_0 > 1$.
- Single epidemic, then disease disappears.
  - Proof?
    Hint: SIR phase portrait indicates that every non-equilibrium solution is a heteroclinic orbit.

The SIR model: Results so far

Biological inferences:

- $\mathcal{R}_0$ is extremely important to estimate in practice!
- Epidemic occurs if and only if $\mathcal{R}_0 > 1$.
- Single epidemic, then disease disappears.
  - Proof?
    Hint: SIR phase portrait indicates that every non-equilibrium solution is a heteroclinic orbit.
- Can prevent epidemic by vaccinating (or otherwise removing) a proportion $1 - \frac{1}{\mathcal{R}_0}$ from the transmission process.

The SIR model: Results so far

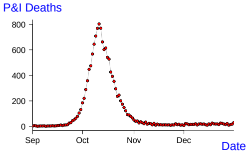
Biological inferences:

- $\mathcal{R}_0$ is extremely important to estimate in practice!
- Epidemic occurs if and only if $\mathcal{R}_0 > 1$.
- Single epidemic, then disease disappears.
  - Proof?
    Hint: SIR phase portrait indicates that every non-equilibrium solution is a heteroclinic orbit.
- Can prevent epidemic by vaccinating (or otherwise removing) a proportion $1 - \frac{1}{\mathcal{R}_0}$ from the transmission process.

Note: It doesn't matter whether we remove people from the susceptible pool by vaccination, isolation, or other means. What matters is the proportion of the population who are removed from the transmission process.

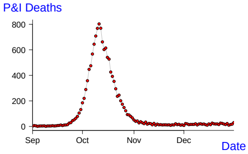
The SIR model: Does it explain our data?

What about 1918 flu in Philadelphia?



The SIR model: Does it explain our data?

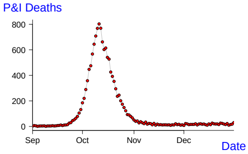
What about 1918 flu in Philadelphia?



- Does the SIR model explain these data?

The SIR model: Does it explain our data?

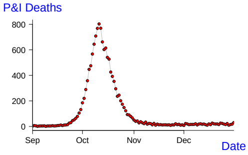
What about 1918 flu in Philadelphia?



- Does the SIR model explain these data?
- Can we fit the SIR model to the P&I time series?

The SIR model: Does it explain our data?

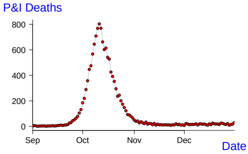
What about 1918 flu in Philadelphia?



- Does the SIR model explain these data?
- Can we fit the SIR model to the P&I time series?
- If so, are our estimated parameter values (for $\mathcal{R}_0$ and $1/\gamma$) biologically reasonable?

The SIR model: Does it explain our data?

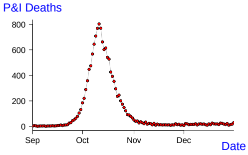
What about 1918 flu in Philadelphia?



- Does the SIR model explain these data?
- Can we fit the SIR model to the P&I time series?
- If so, are our estimated parameter values (for $\mathcal{R}_0$ and $1/\gamma$) biologically reasonable?
- Answers:

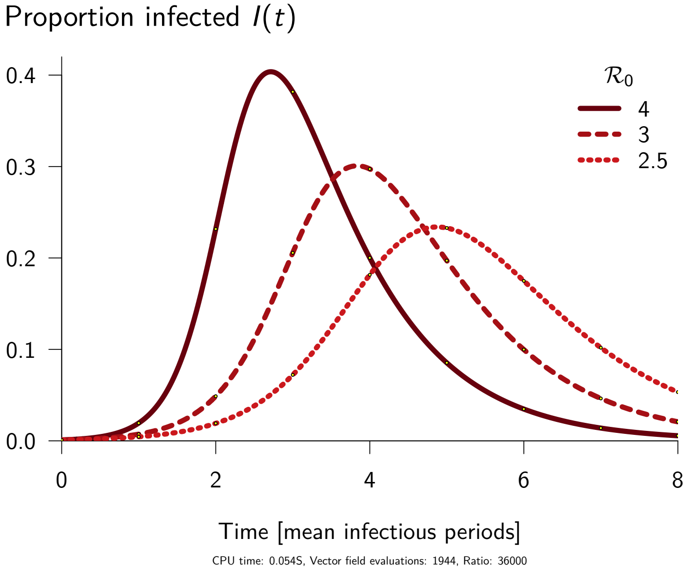
The SIR model: Does it explain our data?

What about 1918 flu in Philadelphia?



- Does the SIR model explain these data?
- Can we fit the SIR model to the P&I time series?
- If so, are our estimated parameter values (for $\mathcal{R}_0$ and $1/\gamma$) biologically reasonable?
- Answers: **Assignment 2...**

The SIR model: How solutions depend on $\mathcal{R}_0$



The SIR model: prevalence *vs.* incidence

The SIR model: prevalence *vs.* incidence

- In the SIR model as we have defined it, prevalence is $I(t)$ and incidence is $$\iota(t) = \beta S(t) I(t)\,,$$

The SIR model: prevalence *vs.* incidence

- In the SIR model as we have defined it, prevalence is $I(t)$ and incidence is
$$\iota(t) = \beta S(t) I(t)\,,$$
so we can compute incidence $\iota(t)$ from solutions to the SIR model

The SIR model: prevalence *vs.* incidence

- In the SIR model as we have defined it, prevalence is $I(t)$ and incidence is
$$\iota(t) = \beta S(t) I(t)\,,$$
so we can compute incidence $\iota(t)$ from solutions to the SIR model, and then compare predicted incidence with observed reports of cases or deaths.

The SIR model: prevalence *vs.* incidence

- In the SIR model as we have defined it, prevalence is $I(t)$ and incidence is
$$\iota(t) = \beta S(t) I(t)\,,$$
so we can compute incidence $\iota(t)$ from solutions to the SIR model, and then compare predicted incidence with observed reports of cases or deaths.
- Could we have derived an equally sensible model starting from the closer-to-observable quantity (incidence ι) ?

The SIR model: prevalence *vs.* incidence

- In the SIR model as we have defined it, prevalence is $I(t)$ and incidence is
$$\iota(t) = \beta S(t) I(t)\,,$$
so we can compute incidence $\iota(t)$ from solutions to the SIR model, and then compare predicted incidence with observed reports of cases or deaths.
- Could we have derived an equally sensible model starting from the closer-to-observable quantity (incidence ι) ?
- The answer is

The SIR model: prevalence *vs.* incidence

- In the SIR model as we have defined it, prevalence is $I(t)$ and incidence is
$$\iota(t) = \beta S(t) I(t)\,,$$
so we can compute incidence $\iota(t)$ from solutions to the SIR model, and then compare predicted incidence with observed reports of cases or deaths.
- Could we have derived an equally sensible model starting from the closer-to-observable quantity (incidence ι) ?
- The answer is YES

The SIR model: prevalence *vs.* incidence

- In the SIR model as we have defined it, prevalence is $I(t)$ and incidence is
$$\iota(t) = \beta S(t) I(t)\,,$$
so we can compute incidence $\iota(t)$ from solutions to the SIR model, and then compare predicted incidence with observed reports of cases or deaths.
- Could we have derived an equally sensible model starting from the closer-to-observable quantity (incidence ι) ?
- The answer is YES,

$$\frac{\mathrm{d}S}{\mathrm{d}t} = -\iota(t)\,, \tag{1a}$$

$$\iota(t) = \mathcal{R}_0\, S(t) \int_0^\infty \iota(t-s)\, g(s)\, \mathrm{d}s\,, \tag{1b}$$

The SIR model: prevalence *vs.* incidence

- In the SIR model as we have defined it, prevalence is $I(t)$ and incidence is
$$\iota(t) = \beta S(t) I(t)\,,$$
so we can compute incidence $\iota(t)$ from solutions to the SIR model, and then compare predicted incidence with observed reports of cases or deaths.
- Could we have derived an equally sensible model starting from the closer-to-observable quantity (incidence ι) ?
- The answer is YES,

$$\frac{\mathrm{d}S}{\mathrm{d}t} = -\iota(t)\,, \tag{1a}$$

$$\iota(t) = \mathcal{R}_0\, S(t) \int_0^\infty \iota(t-s)\, g(s)\, \mathrm{d}s\,, \tag{1b}$$

where $g(s)$ is

The SIR model: prevalence *vs.* incidence

- In the SIR model as we have defined it, prevalence is $I(t)$ and incidence is
$$\iota(t) = \beta S(t) I(t)\,,$$
so we can compute incidence $\iota(t)$ from solutions to the SIR model, and then compare predicted incidence with observed reports of cases or deaths.
- Could we have derived an equally sensible model starting from the closer-to-observable quantity (incidence ι) ?
- The answer is YES,

$$\frac{\mathrm{d}S}{\mathrm{d}t} = -\iota(t)\,, \tag{1a}$$

$$\iota(t) = \mathcal{R}_0\, S(t) \int_0^\infty \iota(t-s)\, g(s)\, \mathrm{d}s\,, \tag{1b}$$

where $g(s)$ is the ***generation interval distribution***.

The SIR model: prevalence *vs.* incidence

- In the SIR model as we have defined it, prevalence is $I(t)$ and incidence is
$$\iota(t) = \beta S(t) I(t)\,,$$
so we can compute incidence $\iota(t)$ from solutions to the SIR model, and then compare predicted incidence with observed reports of cases or deaths.
- Could we have derived an equally sensible model starting from the closer-to-observable quantity (incidence ι) ?
- The answer is YES,
$$\frac{\mathrm{d}S}{\mathrm{d}t} = -\iota(t)\,, \tag{1a}$$
$$\iota(t) = \mathcal{R}_0\, S(t) \int_0^\infty \iota(t-s)\, g(s)\, \mathrm{d}s\,, \tag{1b}$$
where $g(s)$ is the ***generation interval distribution***.
- How do solutions of this integro-differential equation differ from those of the SIR model as we have defined it?

The SIR model: prevalence *vs.* incidence

- In the SIR model as we have defined it, prevalence is $I(t)$ and incidence is
$$\iota(t) = \beta S(t) I(t)\,,$$
so we can compute incidence $\iota(t)$ from solutions to the SIR model, and then compare predicted incidence with observed reports of cases or deaths.
- Could we have derived an equally sensible model starting from the closer-to-observable quantity (incidence ι) ?
- The answer is YES,
$$\frac{\mathrm{d}S}{\mathrm{d}t} = -\iota(t)\,, \tag{1a}$$
$$\iota(t) = \mathcal{R}_0\, S(t) \int_0^\infty \iota(t-s)\, g(s)\, \mathrm{d}s\,, \tag{1b}$$
where $g(s)$ is the ***generation interval distribution***.
- How do solutions of this integro-differential equation differ from those of the SIR model as we have defined it?

If you are curious, see Champredon, Dushoff & Earn 2018.

LaTeX

TeX and LaTeX

TeX and LaTeX

- TeX is a computer-based typesetting system designed and implemented by Donald Knuth in the 1970s.

TeX and LaTeX

- TeX is a computer-based typesetting system designed and implemented by Donald Knuth in the 1970s.
- LaTeX is a particular TeX format written by Leslie Lamport. It has become the *de facto* standard for mathematical typesetting (*e.g.*, books, journal articles, . . .).

TeX and LaTeX

- TeX is a computer-based typesetting system designed and implemented by Donald Knuth in the 1970s.
- LaTeX is a particular TeX format written by Leslie Lamport. It has become the *de facto* standard for mathematical typesetting (*e.g.*, books, journal articles, . . .).
- TeX has played an important role in the evolution of principles of software development.

TeX and LaTeX

- TeX is a computer-based typesetting system designed and implemented by Donald Knuth in the 1970s.
- LaTeX is a particular TeX format written by Leslie Lamport. It has become the *de facto* standard for mathematical typesetting (*e.g.*, books, journal articles, . . .).
- TeX has played an important role in the evolution of principles of software development.
  - Literate programming

TeX and LaTeX

- TeX is a computer-based typesetting system designed and implemented by Donald Knuth in the 1970s.
- LaTeX is a particular TeX format written by Leslie Lamport. It has become the *de facto* standard for mathematical typesetting (*e.g.*, books, journal articles, ...).
- TeX has played an important role in the evolution of principles of software development.
    - Literate programming
    - Reproducible research

TeX and LaTeX

- TeX is a computer-based typesetting system designed and implemented by Donald Knuth in the 1970s.

- LaTeX is a particular TeX format written by Leslie Lamport. It has become the *de facto* standard for mathematical typesetting (*e.g.*, books, journal articles, . . .).

- TeX has played an important role in the evolution of principles of software development.
  - Literate programming
  - Reproducible research

- Immediate goal:

TeX and LaTeX

- TeX is a computer-based typesetting system designed and implemented by Donald Knuth in the 1970s.

- LaTeX is a particular TeX format written by Leslie Lamport. It has become the *de facto* standard for mathematical typesetting (*e.g.*, books, journal articles, . . .).

- TeX has played an important role in the evolution of principles of software development.
  - Literate programming
  - Reproducible research

- Immediate goal: learn enough LaTeX to do Assignment 1.

TeX and LaTeX

- TeX is a computer-based typesetting system designed and implemented by Donald Knuth in the 1970s.
- LaTeX is a particular TeX format written by Leslie Lamport. It has become the *de facto* standard for mathematical typesetting (*e.g.*, books, journal articles, . . .).
- TeX has played an important role in the evolution of principles of software development.
  - Literate programming
  - Reproducible research
- Immediate goal: learn enough LaTeX to do Assignment 1.
- Goal for the term:

TeX and LaTeX

- TeX is a computer-based typesetting system designed and implemented by Donald Knuth in the 1970s.

- LaTeX is a particular TeX format written by Leslie Lamport. It has become the *de facto* standard for mathematical typesetting (*e.g.*, books, journal articles, . . .).

- TeX has played an important role in the evolution of principles of software development.
  - Literate programming
  - Reproducible research

- Immediate goal: learn enough LaTeX to do Assignment 1.

- Goal for the term: become sufficiently competent with LaTeX and R so that the final project can be submitted as a fully reproducible document that "knits" LaTeX and R together.

Getting started

Getting started

- Start `RStudio`

Getting started

- Start `RStudio`
- Work by constructing an R script (see Rexamples.R, accessible from Lecture Schedule).

Getting started

- Start `RStudio`
- Work by constructing an R script (see Rexamples.R, accessible from Lecture Schedule).
- Try some arithmetic and basic plotting.

Getting started

- Start `RStudio`
- Work by constructing an R script (see Rexamples.R, accessible from Lecture Schedule).
- Try some arithmetic and basic plotting.
- Then try to make publication-quality graphs.

Getting started

- Start `RStudio`
- Work by constructing an R script (see Rexamples.R, accessible from Lecture Schedule).
- Try some arithmetic and basic plotting.
- Then try to make publication-quality graphs.
- Other resources:

Getting started

- Start `RStudio`
- Work by constructing an R script (see Rexamples.R, accessible from Lecture Schedule).
- Try some arithmetic and basic plotting.
- Then try to make publication-quality graphs.
- Other resources:
  - Jonathan Dushoff's R intro

Getting started

- Start `RStudio`
- Work by constructing an R script (see Rexamples.R, accessible from Lecture Schedule).
- Try some arithmetic and basic plotting.
- Then try to make publication-quality graphs.
- Other resources:
  - Jonathan Dushoff's R intro
  - Ben Bolker's R intro

Getting started

- Start `RStudio`
- Work by constructing an R script (see Rexamples.R, accessible from Lecture Schedule).
- Try some arithmetic and basic plotting.
- Then try to make publication-quality graphs.
- Other resources:
  - Jonathan Dushoff's R intro
  - Ben Bolker's R intro
  - R Project home page

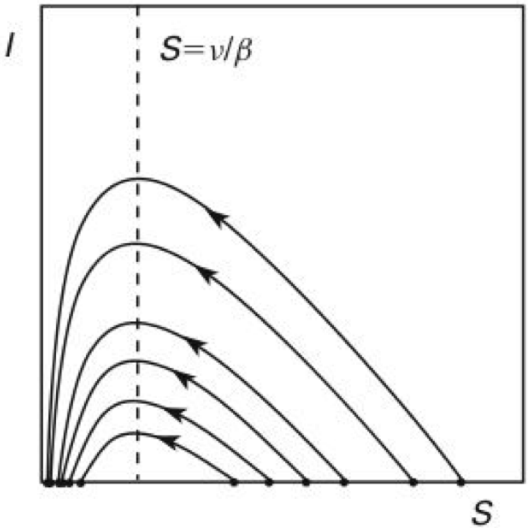
Getting started

- Start `RStudio`
- Work by constructing an R script (see Rexamples.R, accessible from Lecture Schedule).
- Try some arithmetic and basic plotting.
- Then try to make publication-quality graphs.
- Other resources:
  - Jonathan Dushoff's R intro
  - Ben Bolker's R intro
  - R Project home page
  - The official "Introduction to R" by Venables and Smith (html, pdf)

Getting started

- Start `RStudio`
- Work by constructing an R script (see Rexamples.R, accessible from Lecture Schedule).
- Try some arithmetic and basic plotting.
- Then try to make publication-quality graphs.
- Other resources:
  - Jonathan Dushoff's R intro
  - Ben Bolker's R intro
  - R Project home page
  - The official "Introduction to R" by Venables and Smith (html, pdf)
  - Data Camp

Figure 11.2 from HSD* (original from book)



*Hirsch, Smale and Devaney (2013), "Differential equations, dynamical systems, and an introduction to chaos".

Figure 11.2 from HSD (made from scratch in R)

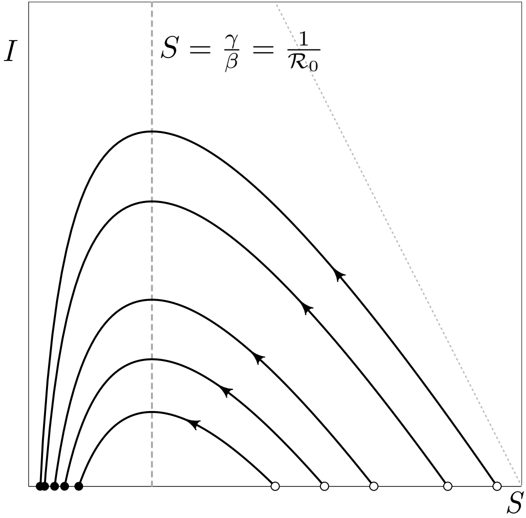


Figure 11.1 from HSD (original from book)

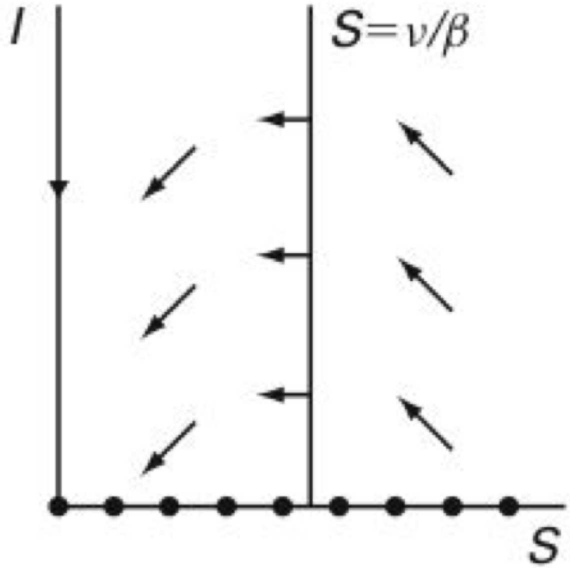


Figure 11.1 from HSD (made from scratch in R)