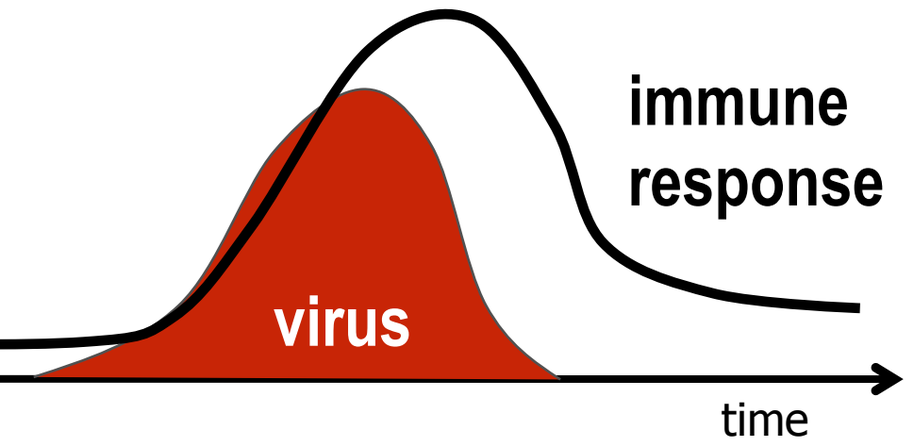
Physics of Life Data Epidemiology

Lect 2: Population dynamics 1

Chiara Poletto
mail: chiara.poletto@unipd.it
web: chiara-poletto.github.io
bsky: @chpoletto.bsky.social

the 3 ingredients of an epidemic



disease natural history

+

transmission

directly transmitted
- airborne
- droplets
- physical contact (hand shake)
- sexual contact

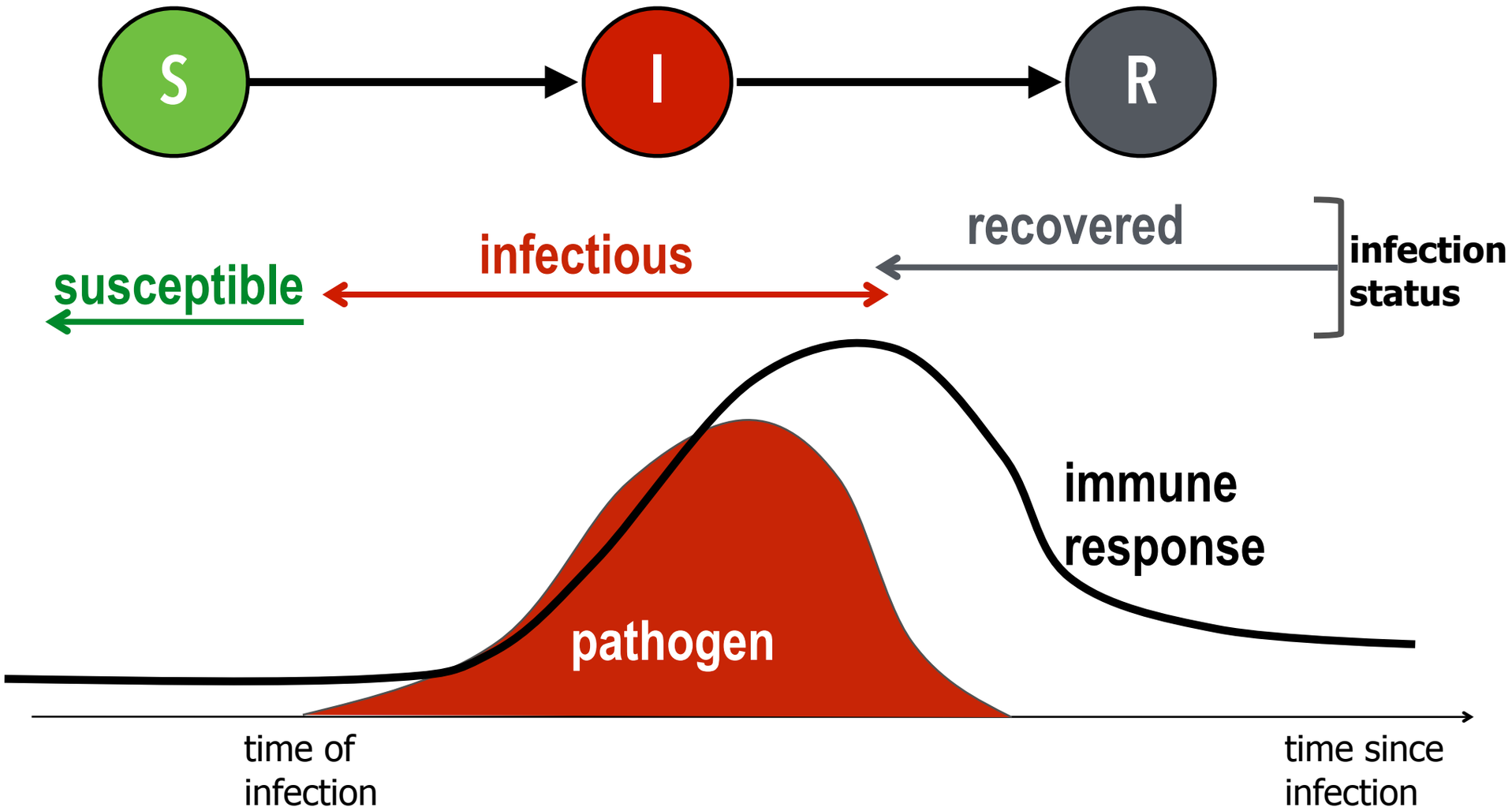
non directly transmitted
- vector-borne
- food-born
- fomites

+

contact patterns

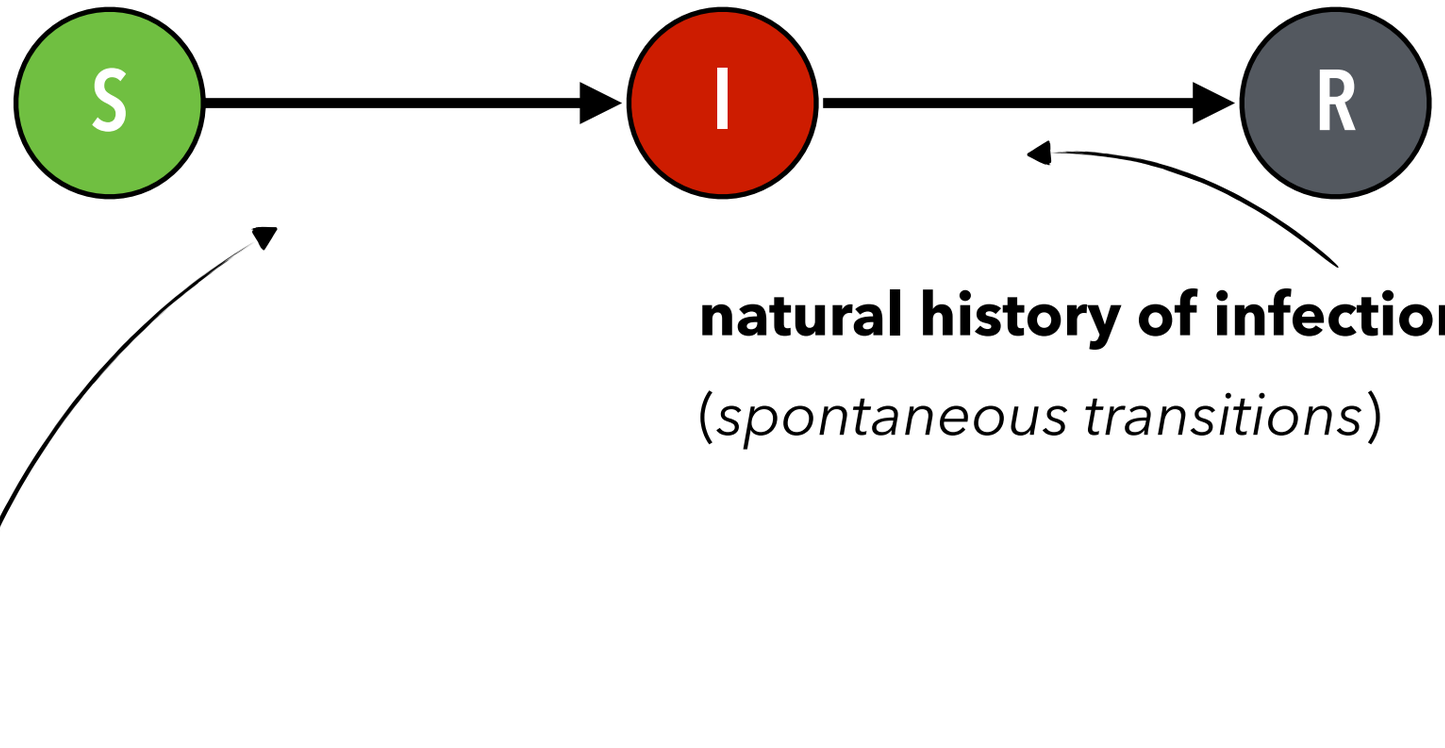
a time-varying spatially-explicit network

modelling disease natural history



[Keeling & Rohani, Modeling Infectious Diseases (2008)]

modelling transitions across disease stages



natural history of infection

(*spontaneous transitions*)

contagion

coupling with the infectious status of other individuals' (*mediated transition*)

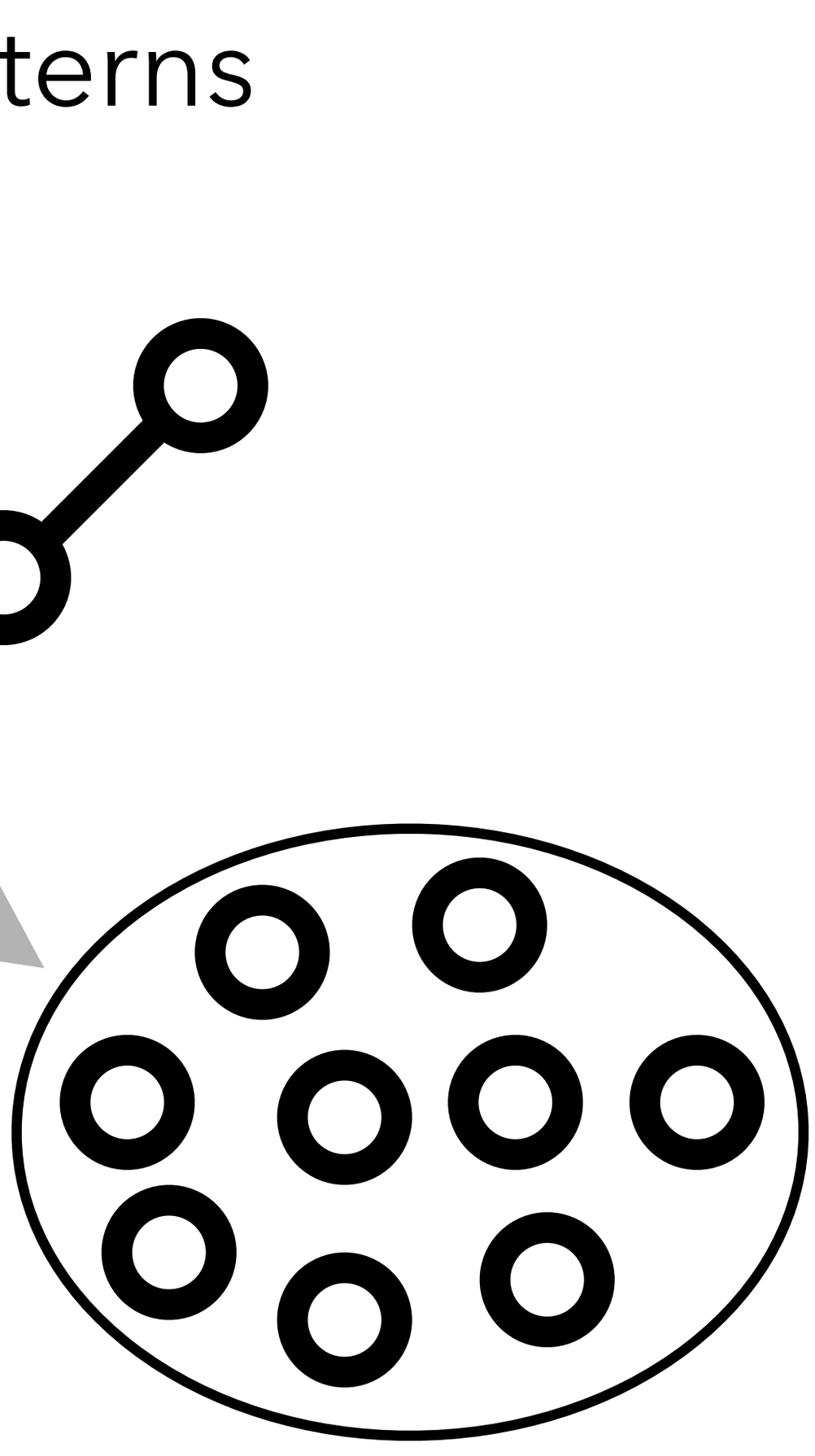
[Keeling & Rohani, Modeling Infectious Diseases (2008)]

modelling transmission

if a susceptible enter in contact with an infectious individual he/she can contract the infection with a probability β per unit time

[Keeling & Rohani, Modeling Infectious Diseases (2008)]

modelling contact patterns

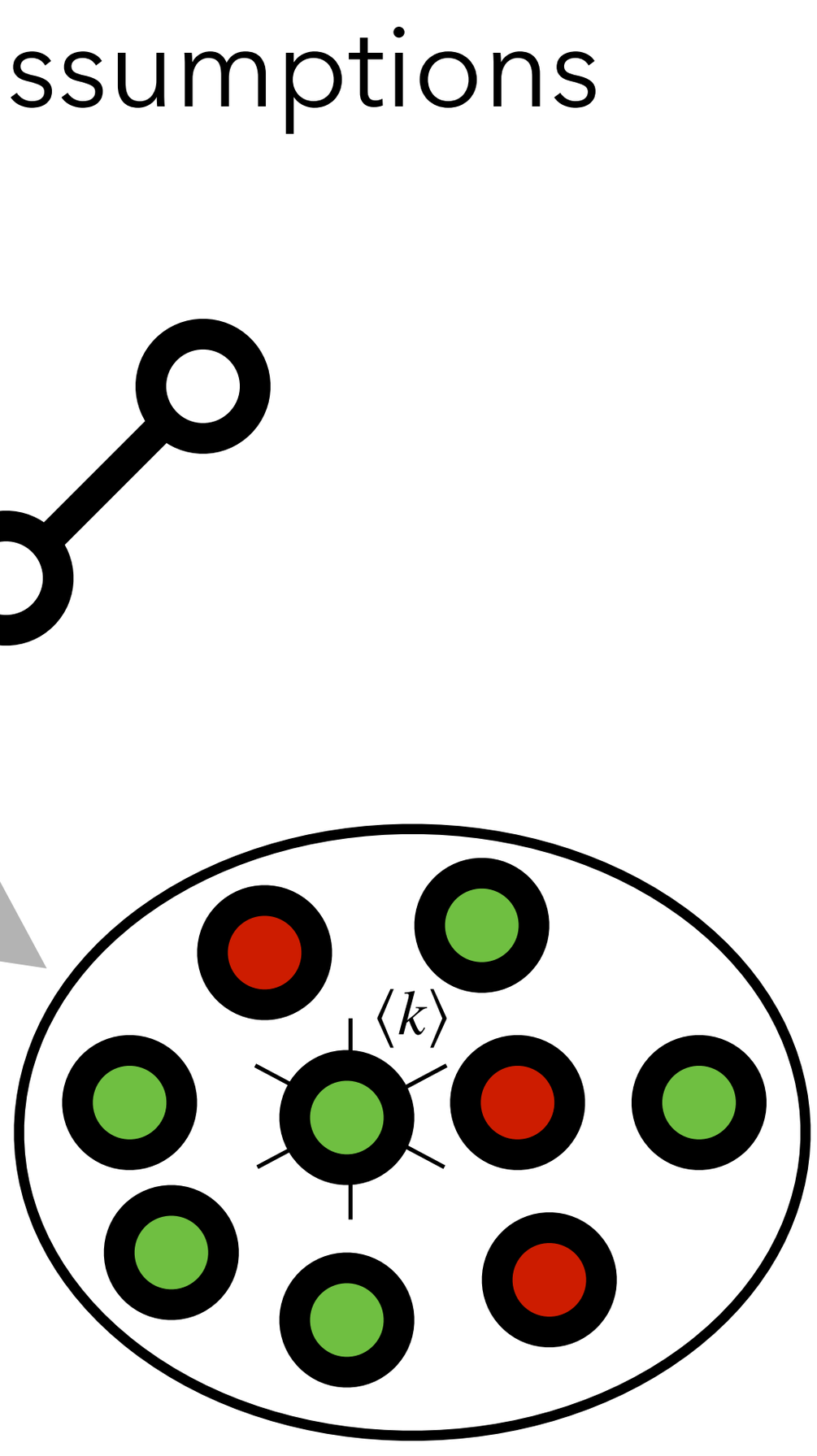


homogenous mixing assumption

- All individuals behave equally
- All individuals have the same number of contacts $\langle k \rangle$

Unrealistic, especially for large populations (i.e. regions/countries) but easier math

combining the three assumptions

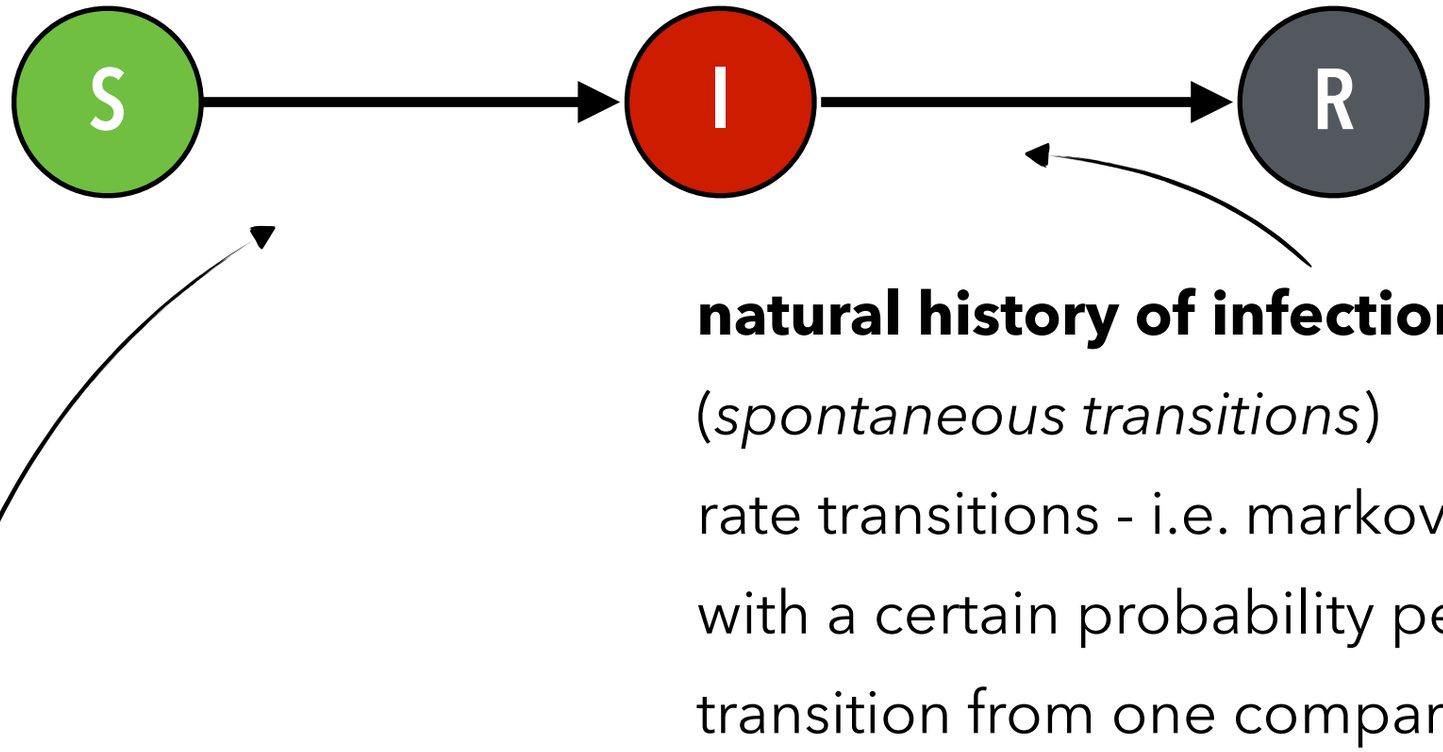


Force of infection=

contact behaviour **X** transmission

$$\langle k \rangle \frac{I}{N} \times \beta$$

combining the three assumptions



natural history of infection

(*spontaneous transitions*)

rate transitions - i.e. markovian transitions

with a certain probability per unit of time I can

transition from one compartment to another

contagion

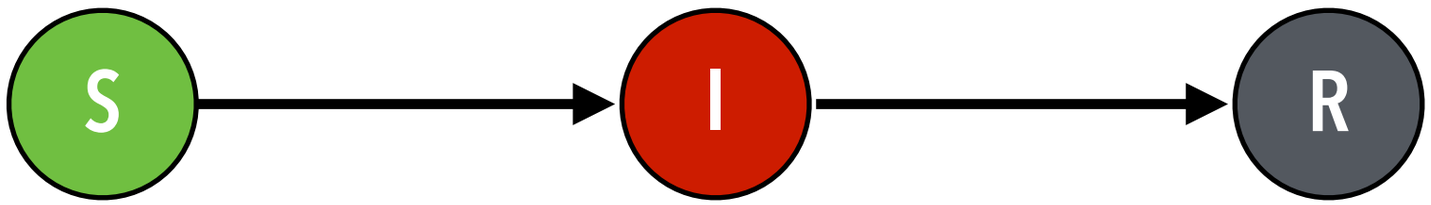
coupling with the infectious status of other individuals' (*mediated transition*)

Force of infection: probability per unit time for a susceptible individual to acquire the infection

this is given by $\langle k \rangle \frac{I}{N} \times \beta$

[Keeling & Rohani, Modeling Infectious Diseases (2008)]

population dynamics models (compartmental models)

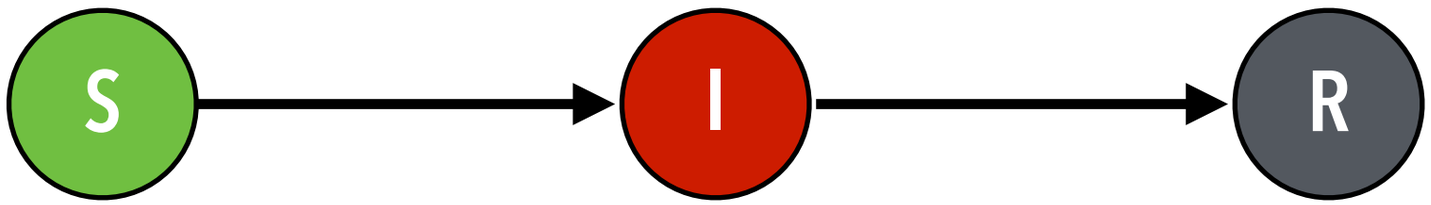


Population dynamics: the mathematics that deals with the variation in time and space of population size and density for one or more species (e.g. Lotka–Volterra model)

Populations are the individuals in each infection stage (also called compartments). Variables represent the occupation number of each stage/compartment

S,I,R = number of individuals in the population that are susceptible, infectious, recovered, respectively

population dynamics models (compartmental models)



S,I,R = number of individuals in the population that are susceptible, infectious, recovered, respectively

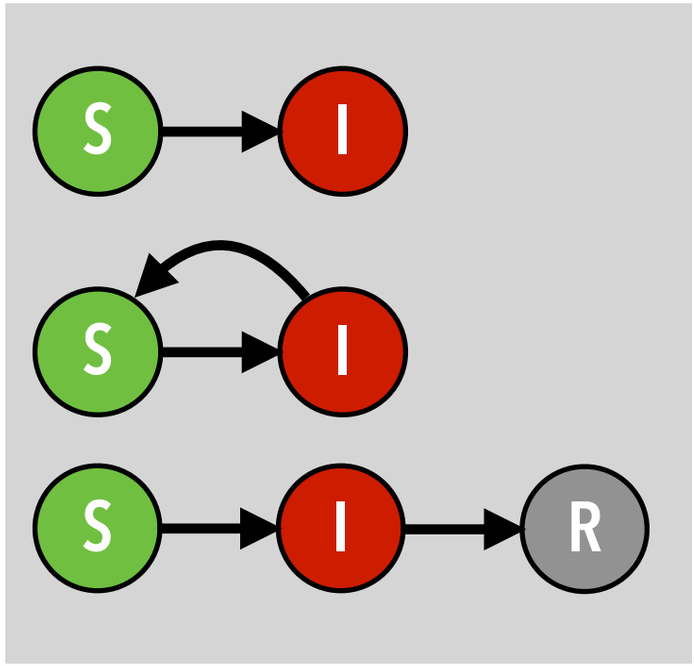
Maintaining the rules of the process the same we can have that:

- Occupation of compartments can be represented as discrete or continuous variables
- time can be discrete or continuous
- The process can be stochastic or deterministic

continuous time, continuous variable, deterministic -> Ordinary differential equations

discrete time, discrete variable, stochastic -> Binomial chains

compartmental models



continuous time, continuous variable, deterministic

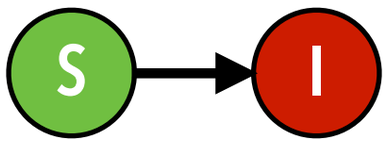
[Keeling & Rohani, Modeling Infectious Diseases (2008)]

Notation

Prevalence number of infectious at time t, i.e. occupation number of the compartment I

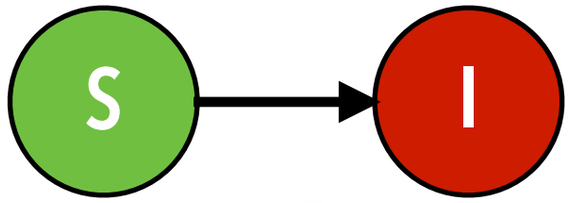
Incidence number of new infectious cases at time t. Incidence is a central quantity in epidemiology since it is the one that can be measured

Attack rate (or size) in a given time window: total number of cases in the time window, i.e. cumulative of incidence during that window


S
I

SI Model

- Infected stay infected forever
- Good approximation for chronic infections (Tuberculosis, HIV, Hepatitis)
- **Note:** you never remain infectious forever, e.g. even for chronic infections you can cease to be infectious thanks to treatment. Still, the SI is the preferred approximation in many cases



$$\beta\langle k\rangle\frac{I}{N} =$$

Force of Infection (FOI)

reaction rules:

$$S + I \xrightarrow{\beta} I + I$$

SI Model

Ordinary differential equations

using occupation numbers as variables

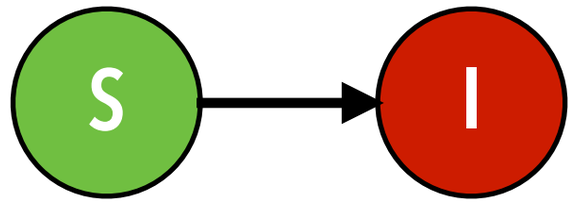
$$\frac{dS}{dt} = -\beta\langle k\rangle\frac{S\,I}{N}$$

$$\frac{dI}{dt} = \beta\langle k\rangle\frac{S\,I}{N}$$

using densities as variables

$$\frac{ds}{dt} = -\beta\langle k\rangle si$$

$$\frac{di}{dt} = \beta\langle k\rangle si$$



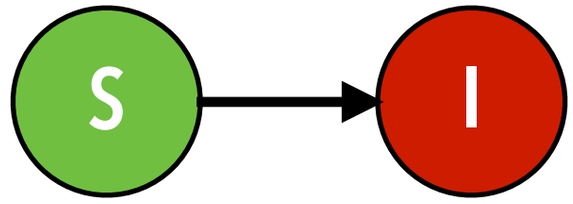
Assumptions:

- N whole population
- $S, I > 0$
- $S + I = N =$ const (close population, i.e. we neglect vital dynamics and immigration)

Definitions:

- S, I Capital letters for integer numbers (individuals)
- $s = S/N$, $i = I/N$ for densities
- **prevalence: I**
- **incidence:** $\beta\langle k\rangle\frac{S\,I}{N}$

SI Model



Definitions:

- i_0 number of infectious at t_0 (seeds of epidemic)

Solution

Since $s + i = 1$ we only have one equation

$$\frac{di}{dt} = \beta i(1 - i)$$

$$\frac{1}{\beta i(1-i)} di = dt \rightarrow \frac{1}{\beta(1-i)} di + \frac{1}{\beta i} di = dt$$

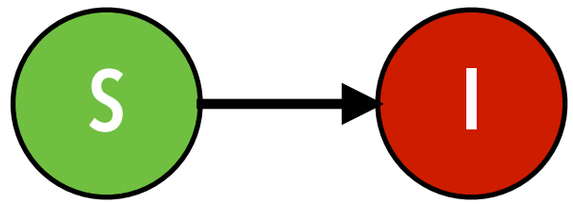
Integrating both sides

$$\frac{1}{\beta(1-i)} di + \frac{1}{\beta i} di = dt$$

$$\frac{i}{1-i} = e^{\beta(t+C)} = Ae^{\beta t}, \text{ with } A = i_0/(1 - i_0)$$

$$i(t) = \frac{i_0 e^{\beta t}}{1 - i_0 + i_0 e^{\beta t}}$$

SI Model

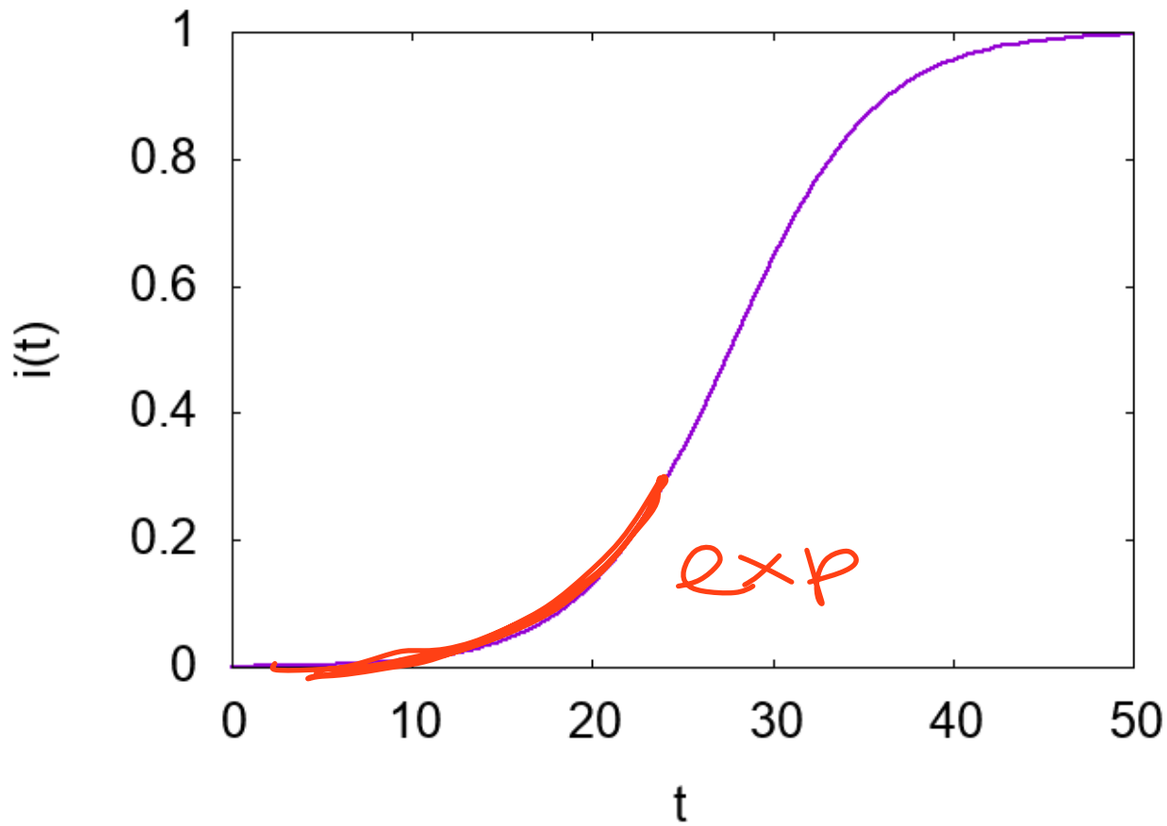


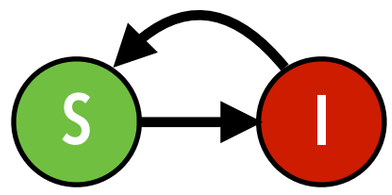
Solution: logistic/sigmoid

$$i(t) = \frac{i_0 e^{\beta t}}{1 - i_0 + i_0 e^{\beta t}}$$

Behavior:

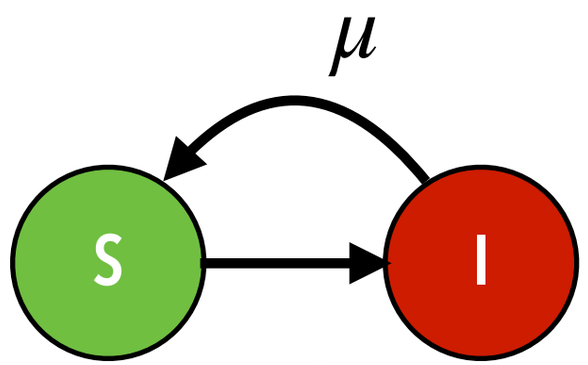
- Early phases: exponential growth governed by β. Higher the β, faster is the growth in cases
- Always saturates at 1 (two steady states, $i = 0$ or $s = 0$)




S
I

SIS Model

- For diseases that approx do not confer immunity:
  - Common Cold (rhinovirus, RSV), HPV (papilloma virus)
- μ: recovering rate $\rightarrow \mu = \tau^{-1}$ i.e. inverse of the infectious period



$$\beta\langle k\rangle\frac{I}{N} =$$

Force of Infection (FOI)

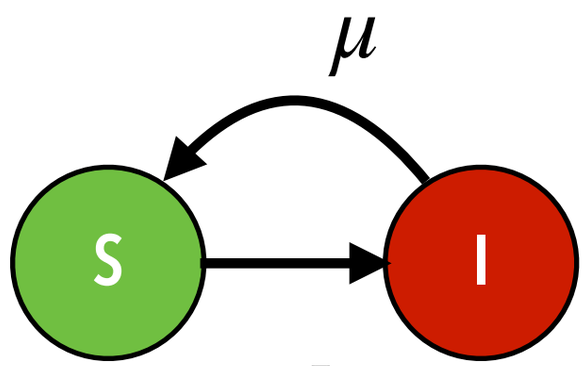
reaction rules:

$S + I \xrightarrow{\beta} I + I$ (mediated transition)

$I \xrightarrow{\mu} S$ (spontaneous transition)

SIS Model

- For diseases that do not confer immunity:
  - Common Cold (rhinovirus, RSV), HPV (papilloma virus)
- μ: recovering rate $\rightarrow \mu = \tau^{-1}$ i.e. inverse of the infectious period



$$\beta\langle k\rangle\frac{I}{N} =$$

Force of Infection (FOI)

reaction rules:

$S + I \xrightarrow{\beta} I + I$ (mediated transition)

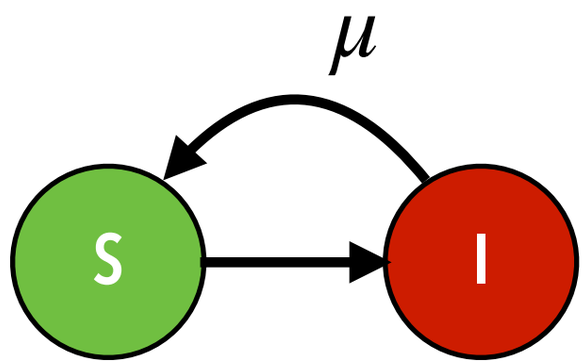
$I \xrightarrow{\mu} S$ (spontaneous transition)

Ordinary differential equations

$$\frac{ds}{dt} = -\beta\langle k\rangle si + \mu i$$

$$\frac{di}{dt} = \beta\langle k\rangle si - \mu i$$

SIS Model



Solution:

$$\frac{di}{dt} = \beta\langle k\rangle(1-i)i - \mu i$$

following a similar reasoning to the SI

$$i(t) = i_0 \frac{(\beta\langle k\rangle - \mu)e^{(\beta\langle k\rangle - \mu)t}}{\beta\langle k\rangle - \mu + \beta\langle k\rangle i_0 e^{(\beta\langle k\rangle - \mu)t}}$$

Ordinary differential equations

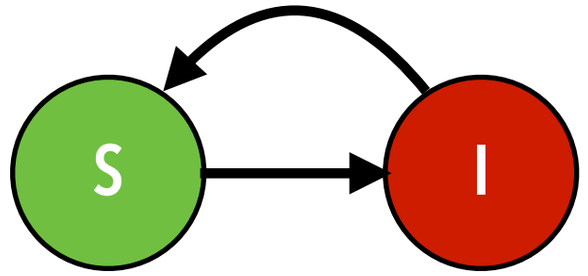
$$\frac{ds}{dt} = -\beta\langle k\rangle si + \mu i$$

$$\frac{di}{dt} = \beta\langle k\rangle si - \mu i$$

i_0 number of infectious at t_0

(seeds of epidemic)

SIS Model



Steady state: $\frac{di}{dt} = 0$

$\beta\langle k\rangle(1-i)i - \mu i = 0$

$i(\beta\langle k\rangle(1-i) - \mu) = 0 \rightarrow i = 0,\ i = \frac{\beta\langle k\rangle - \mu}{\beta\langle k\rangle}$

if $\beta < \beta_c = \frac{\mu}{\langle k\rangle}$:

$i = 0$ stable

$i = \frac{\beta\langle k\rangle - \mu}{\beta\langle k\rangle}$ unfeasible

if $\beta > \beta_c = \frac{\mu}{\langle k\rangle}$:

$i = 0$ unstable

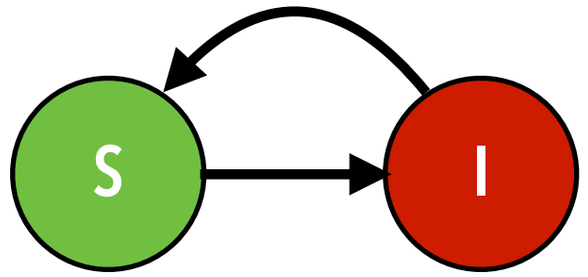
$i = \frac{\beta\langle k\rangle - \mu}{\beta\langle k\rangle}$ feasible

Ordinary differential equations

$$\frac{ds}{dt} = -\beta\langle k\rangle si + \mu i$$

$$\frac{di}{dt} = \beta\langle k\rangle si - \mu i$$

SIS Model



Steady state: $\frac{di}{dt} = 0$

$\beta\langle k\rangle(1-i)i - \mu i = 0$

$i(\beta\langle k\rangle(1-i) - \mu) = 0 \rightarrow i = 0,\ i = \frac{\beta\langle k\rangle - \mu}{\beta\langle k\rangle}$

if $\beta < \beta_c = \frac{\mu}{\langle k\rangle}$:

$i = 0$ stable

$i = \frac{\beta\langle k\rangle - \mu}{\beta\langle k\rangle}$ unfeasible

disease-free equilibrium,

epidemic extinction

if $\beta > \beta_c = \frac{\mu}{\langle k\rangle}$:

$i = 0$ unstable

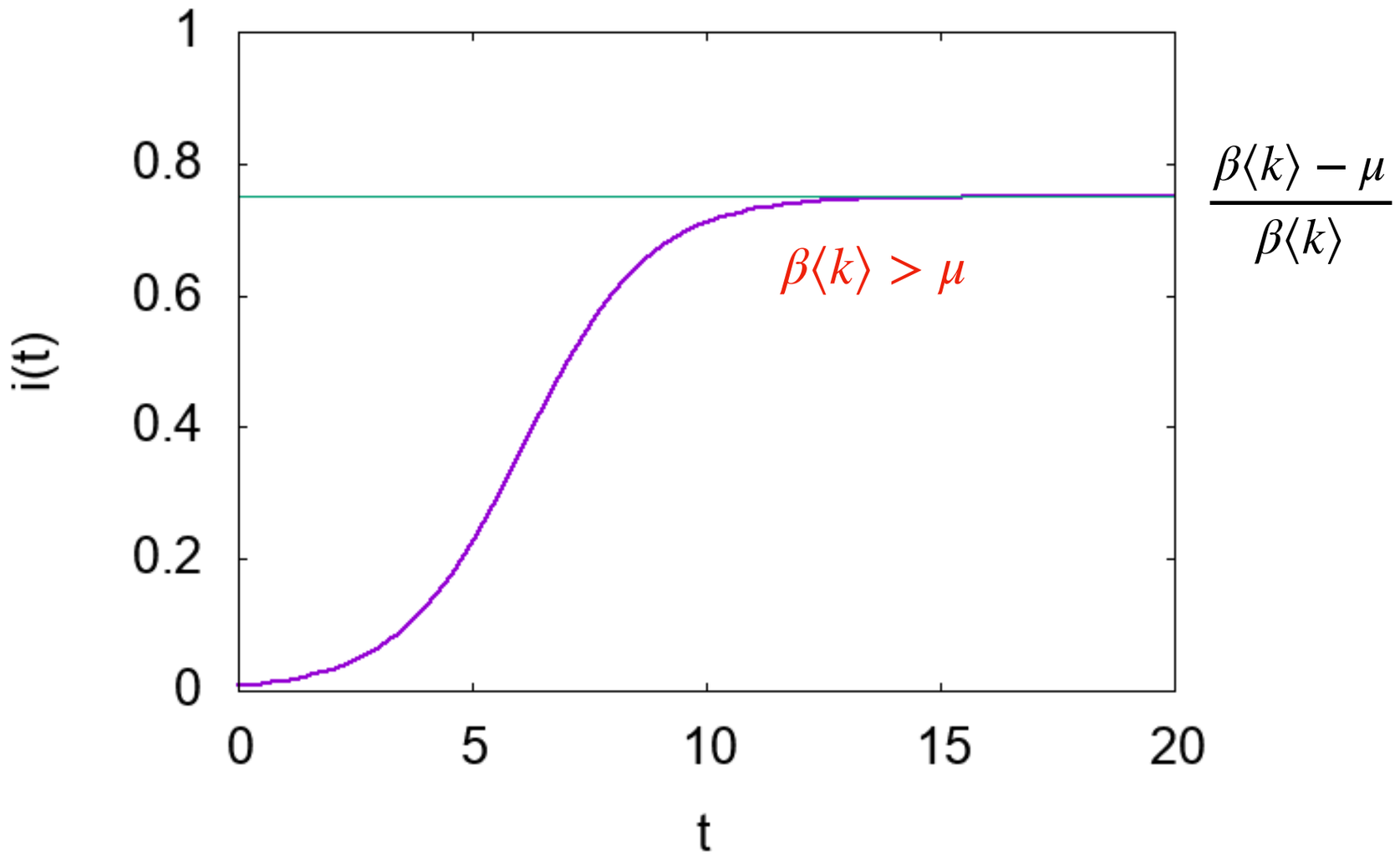
$i = \frac{\beta\langle k\rangle - \mu}{\beta\langle k\rangle}$ feasible

active stationary state, endemic

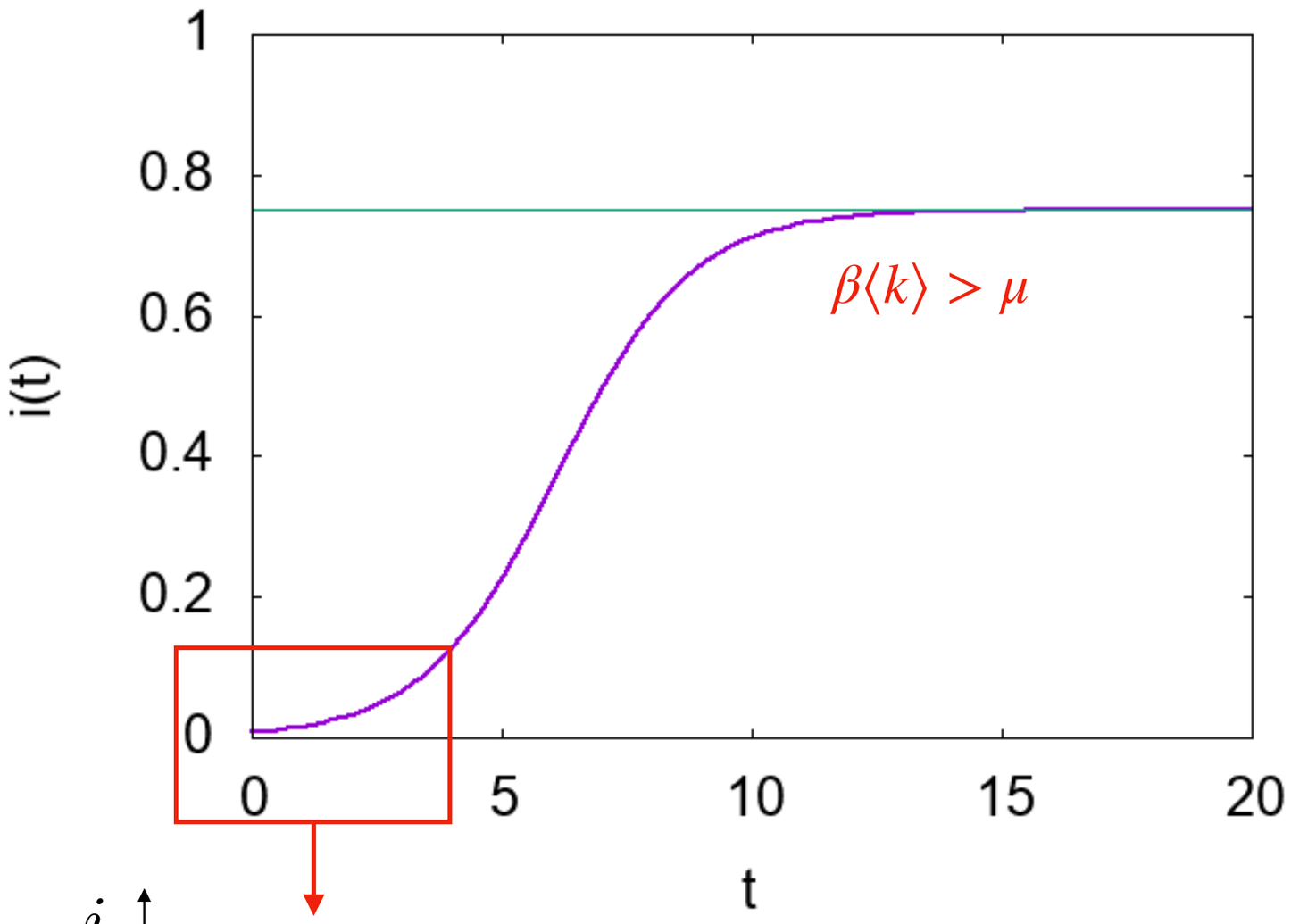
equilibrium,

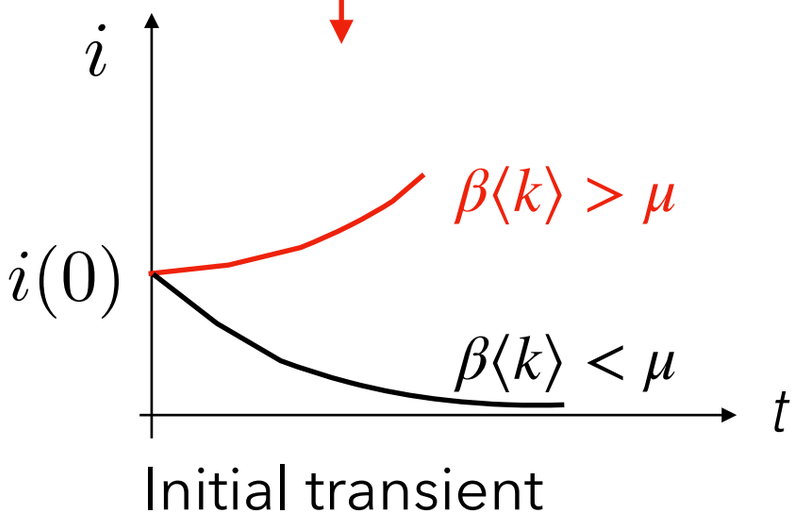
new recovered = # new infections

SIS Model


1
0.8
0.6
0.4
0.2
0
i(t)
0
5
10
15
20
t
$\beta\langle k\rangle > \mu$
$\frac{\beta\langle k\rangle - \mu}{\beta\langle k\rangle}$

SIS Model





Initial transient

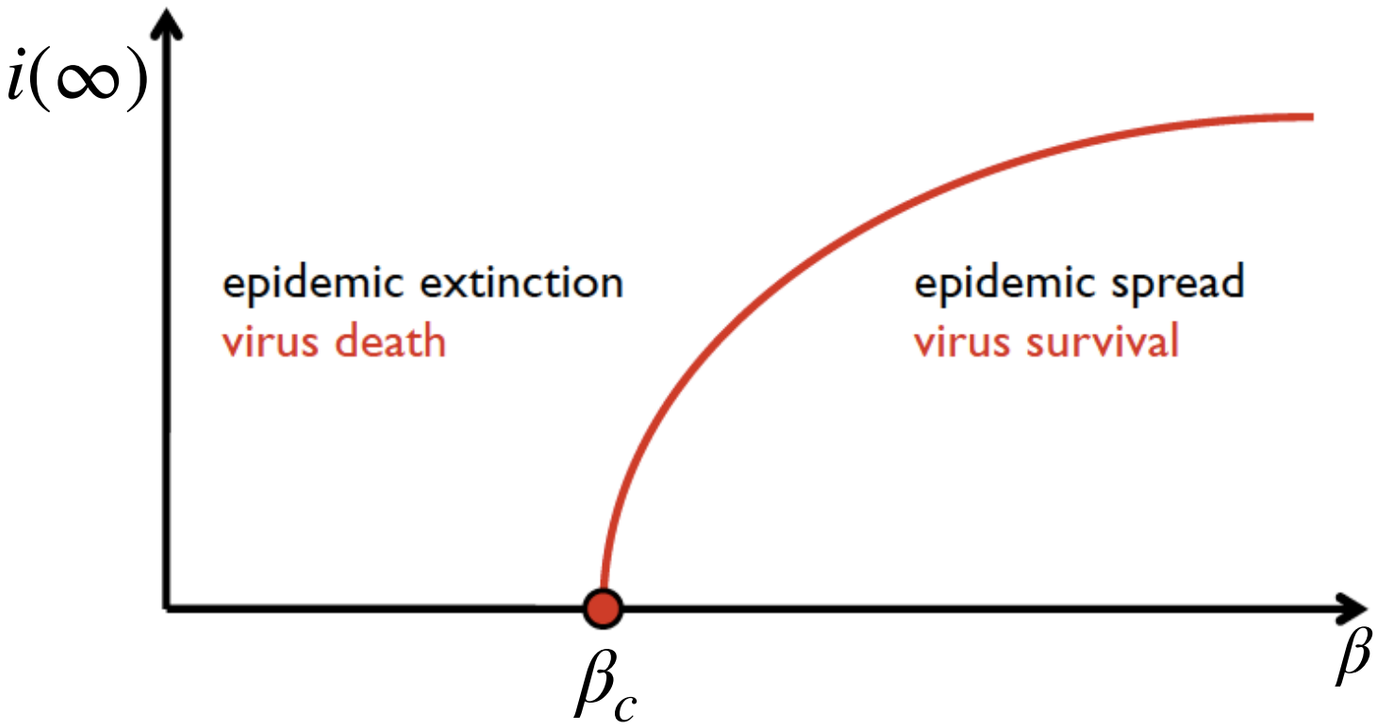
Early stages: $s \simeq 1$ and $i \ll 1$

$$\frac{di}{dt} = \beta\langle k\rangle si - \mu i \simeq \beta\langle k\rangle i - \mu i$$

$$i(t) \simeq i_0 e^{(\beta\langle k\rangle - \mu)t}$$

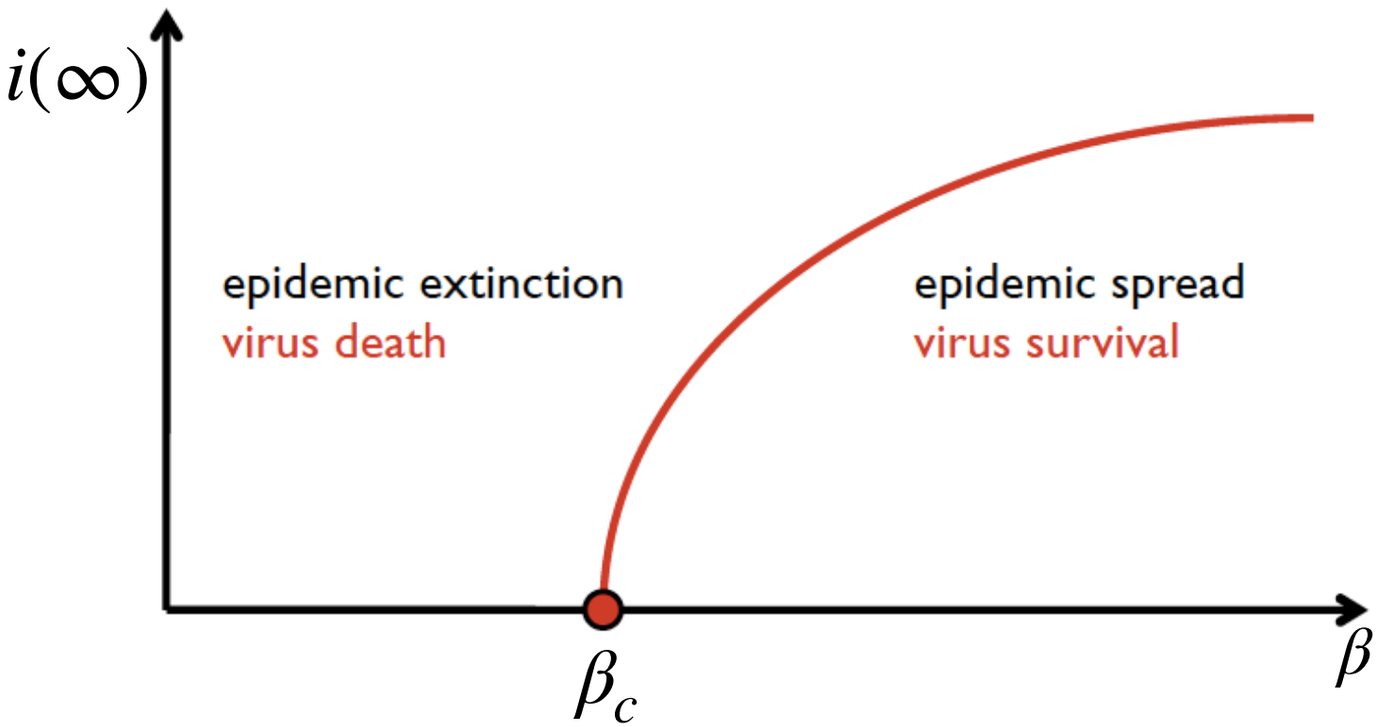
Epidemic threshold

$\beta > \beta_c = \frac{\mu}{\langle k \rangle}$: **Epidemic threshold**, i.e. minimum value of the infection probability for which the disease survives



Epidemic threshold

$\beta > \beta_c = \frac{\mu}{\langle k \rangle}$: **Epidemic threshold**, i.e. minimum value of the infection probability for which the disease survives



Similarities with statistical mechanics

phase transition between

Absorbing = extinction

Active (endemic) = survival

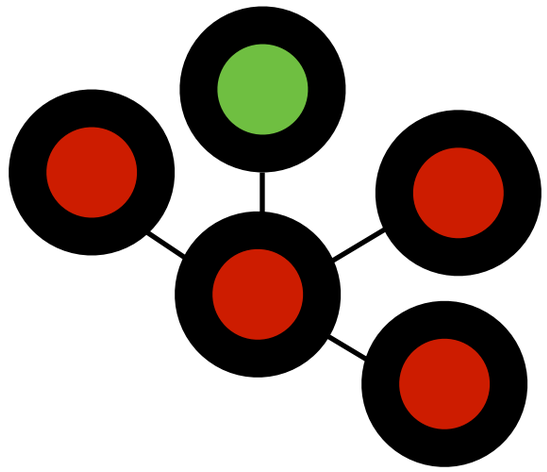
β: control parameter

$i(\infty)$: order parameter

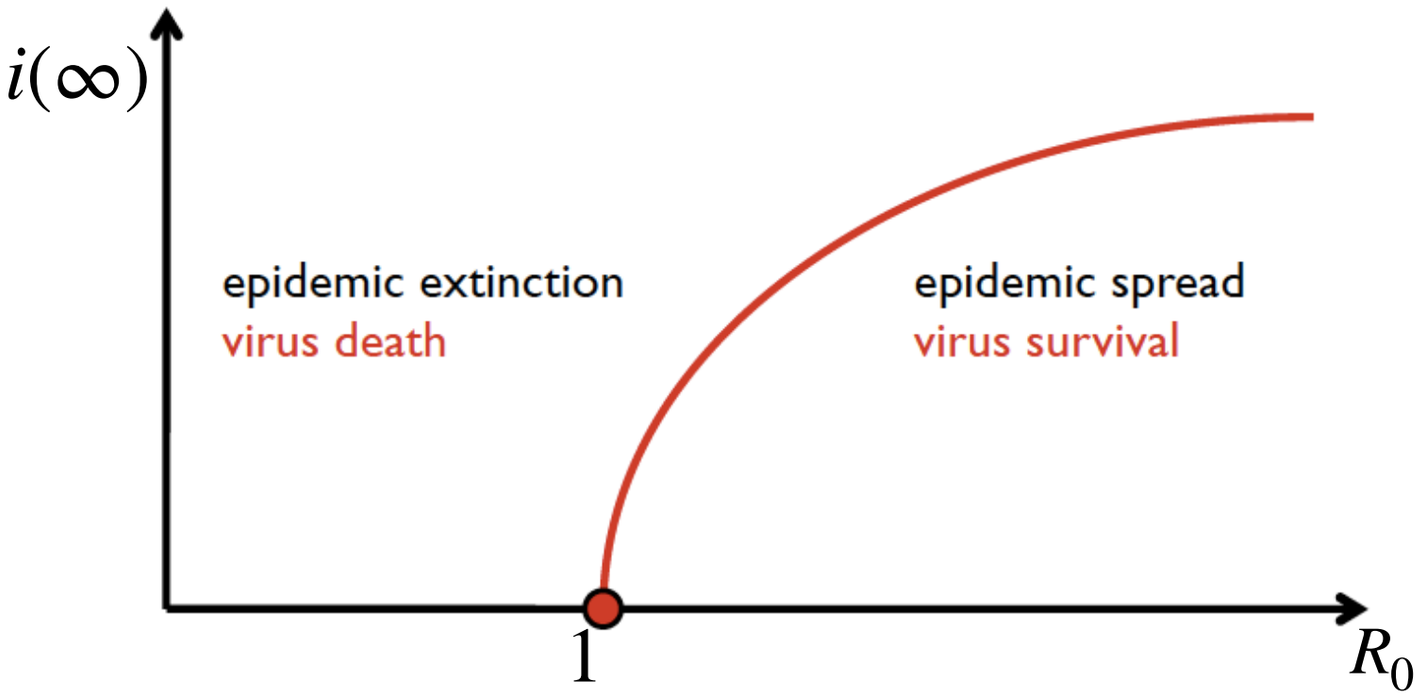
Second order phase-transition,

(critical behaviour, critical exponents)

Epidemic threshold



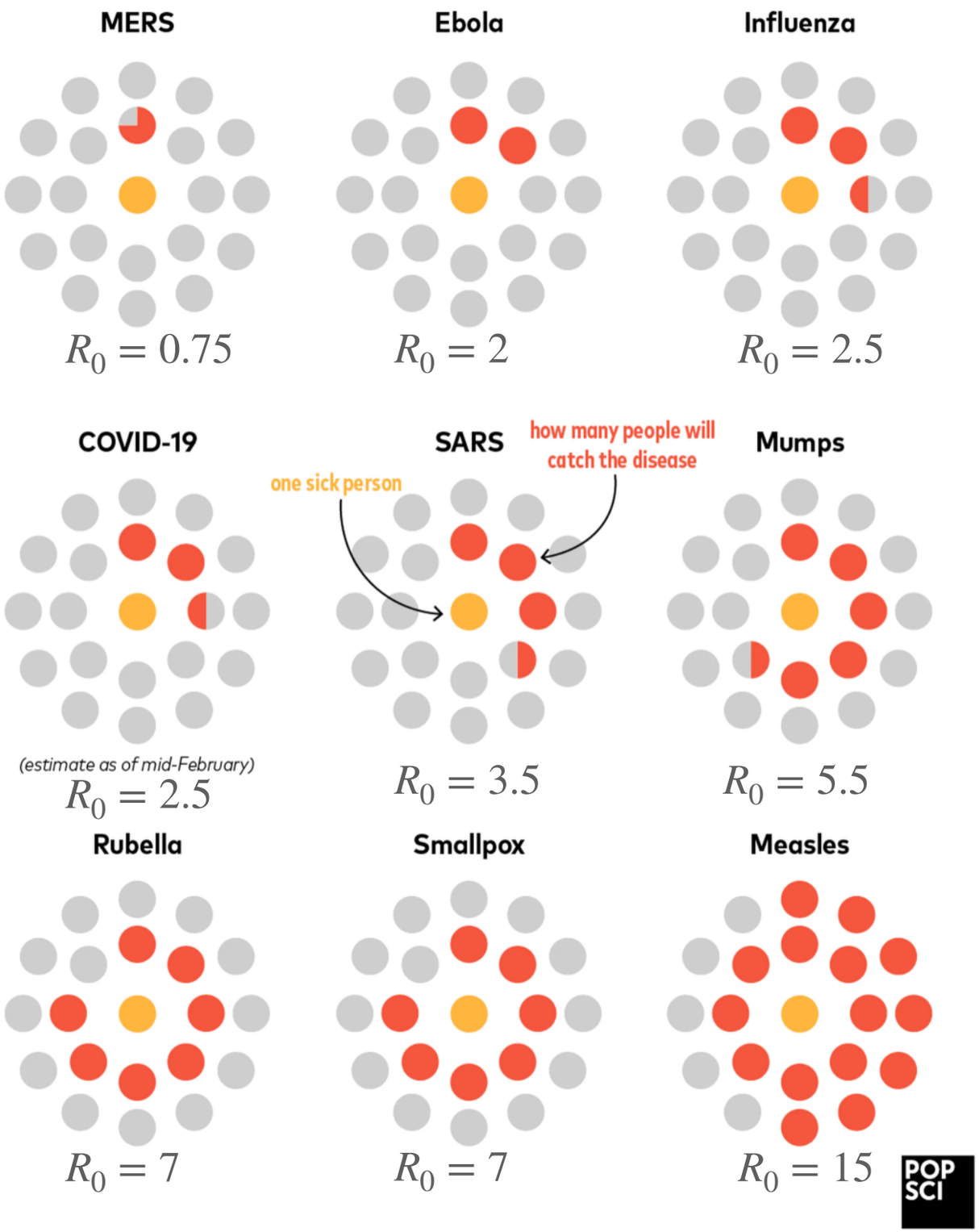
$$\beta > \beta_c = \frac{\mu}{\langle k \rangle}, \text{ i.e. } \frac{\beta \langle k \rangle}{\mu} > 1, \text{ i.e. } R_0 > 1$$



$$R_0 = \frac{\beta \langle k \rangle}{\mu} = \beta \langle k \rangle \tau$$

basic reproductive ratio: number of cases generated by an infectious individual in a fully susceptible population before he/she recovers

R_0: key concept in epidemiology



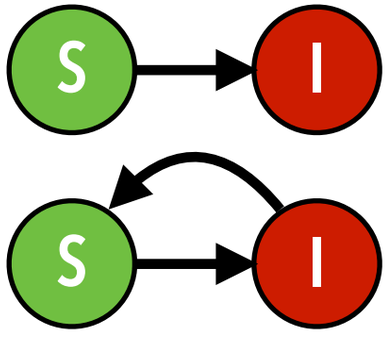
R0 values for well-known diseases. *Sara Chodosh*

R_0 quantify the transmission potential (the *strength*) of an epidemic. It tells us if we are going to have an active epidemic state and how big the epidemic state will be

$$i = \frac{\beta\langle k\rangle - \mu}{\beta\langle k\rangle} = 1 - \frac{1}{R_0}$$

R_0 is a property of an epidemic i.e. resulting from both pathogen and population characteristics. Same pathogen can have different R_0 in different populations

SI, SIS models



Epidemic expansion

Endemic circulation

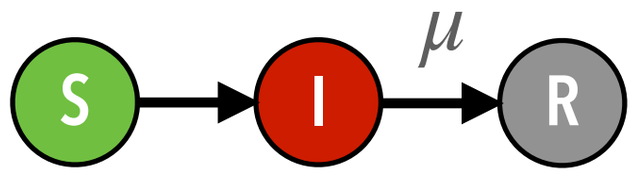
stylised processes, more amenable to analytical calculations. Used in practice in two extreme cases

- in mathematical and physics studies, e.g. networks studies
- for modelling highly complex infections (e.g. AIDS, Malaria)

i.e. Kermack and McKendrick model

SIR Model

- Basic model for long lasting immunity (deaths)
  - Measles, Chickenpox, etc.
  - SIR can be used as long as duration of immunity >> duration of the infectious period (e.g flu, COVID-19 with duration of infection ~days and duration of immunity ~months/ years)
    - most used, it describes an epidemic dynamics (as counterposed to an endemic dynamics)
- μ: recovering rate $\rightarrow \mu = \tau^{-1}$ i.e. inverse of the infectious period



$$\beta\langle k\rangle\frac{I}{N} =$$

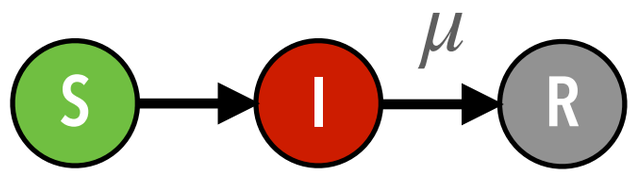
Force of Infection (FOI)

reaction rules:

$S + I \xrightarrow{\beta} I + I$ (mediated transition)

$I \xrightarrow{\mu} R$ (spontaneous transition)

SIR Model



$$\beta\langle k\rangle\frac{I}{N} =$$

Force of Infection (FOI)

Ordinary differential equations

using occupation numbers as variables

$$\frac{dS}{dt} = -\beta\langle k\rangle\frac{S\,I}{N}$$

$$\frac{dI}{dt} = \beta\langle k\rangle\frac{S\,I}{N} - \mu I$$

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

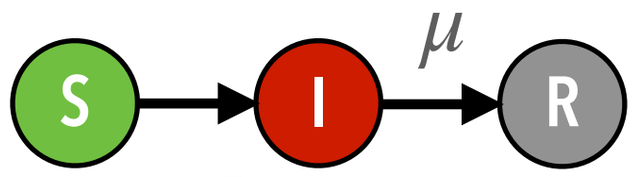
using densities numbers as variables

$$\frac{ds}{dt} = -\beta\langle k\rangle si$$

$$\frac{di}{dt} = \beta\langle k\rangle si - \mu i$$

$$\frac{dr}{dt} = \mu i$$

SIR Model



$$\beta\langle k\rangle\frac{I}{N} =$$

Force of Infection (FOI)

Initial stage: if $s_0 \simeq 1$, $r_0 = 0$ and $i_0 \ll 1$ then SIR $\simeq$ SIS

$$\frac{di}{dt} = \beta\langle k\rangle si - \mu i \simeq \beta\langle k\rangle i - \mu i$$

$$i(t) \simeq i_0 e^{(\beta\langle k\rangle - \mu)t}$$

$R_0 = \beta\langle k\rangle\tau$>1 → same threshold behaviour than SIS

SIR Model

$$\frac{ds}{dt} = -\beta\langle k\rangle si$$
$$\frac{di}{dt} = \beta\langle k\rangle si - \mu i$$
$$\frac{dr}{dt} = \mu i$$

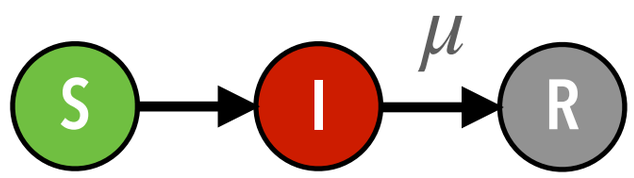
diving ds by dr we get

$$\frac{ds}{dr} = \frac{-\beta\langle k\rangle s}{\mu}$$

Integrating with respect to r and assuming

$r_0 = 0$, we get:

$$s(t) = s_0 e^{-r(t)\frac{\beta\langle k\rangle}{\mu}}$$



$$\beta\langle k\rangle\frac{I}{N} =$$

Force of Infection (FOI)

SIR Model

$$\frac{ds}{dt} = -\beta\langle k\rangle si$$
$$\frac{di}{dt} = \beta\langle k\rangle si - \mu i$$
$$\frac{dr}{dt} = \mu i$$

diving ds by dr we get

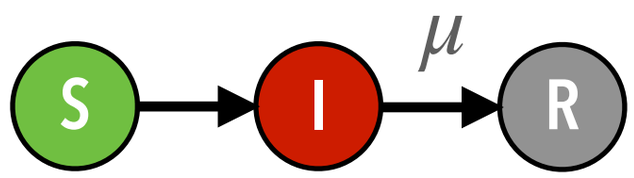
$$\frac{ds}{dr} = \frac{-\beta\langle k\rangle s}{\mu}$$

Integrating with respect to r and assuming

$r_0 = 0$, we get:

$$s(t) = s_0 e^{-r(t)\frac{\beta\langle k\rangle}{\mu}}$$

for $t \to \infty$, $i(t) \to 0 \Rightarrow s(\infty) = 1 - r(\infty)$



$$\beta\langle k\rangle\frac{I}{N} =$$

Force of Infection (FOI)

SIR Model

$$\frac{ds}{dt} = -\beta\langle k\rangle si$$
$$\frac{di}{dt} = \beta\langle k\rangle si - \mu i$$
$$\frac{dr}{dt} = \mu i$$

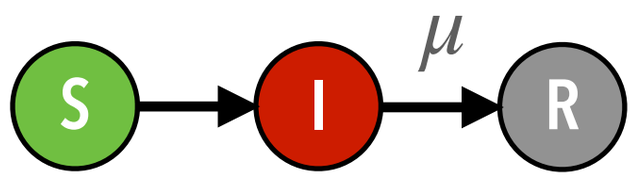
diving ds by dr we get

$$\frac{ds}{dr} = \frac{-\beta\langle k\rangle s}{\mu}$$

Integrating with respect to r and assuming

$r_0 = 0$, we get:

$$1 - r(\infty) = s_0 e^{-r(\infty)\frac{\beta\langle k\rangle}{\mu}}$$



$$\beta\langle k\rangle\frac{I}{N} =$$

Force of Infection (FOI)

SIR Model

$$\frac{ds}{dt} = -\beta\langle k\rangle si$$
$$\frac{di}{dt} = \beta\langle k\rangle si - \mu i$$
$$\frac{dr}{dt} = \mu i$$

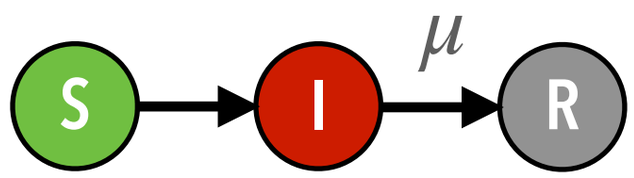
diving ds by dr we get

$$\frac{ds}{dr} = \frac{-\beta\langle k\rangle s}{\mu}$$

Integrating with respect to r and assuming

$r_0 = 0$, we get:

$$1 - r(\infty) = s_0 e^{-r(\infty)R_0}$$



$$\beta\langle k\rangle \frac{I}{N} =$$

Force of Infection (FOI)

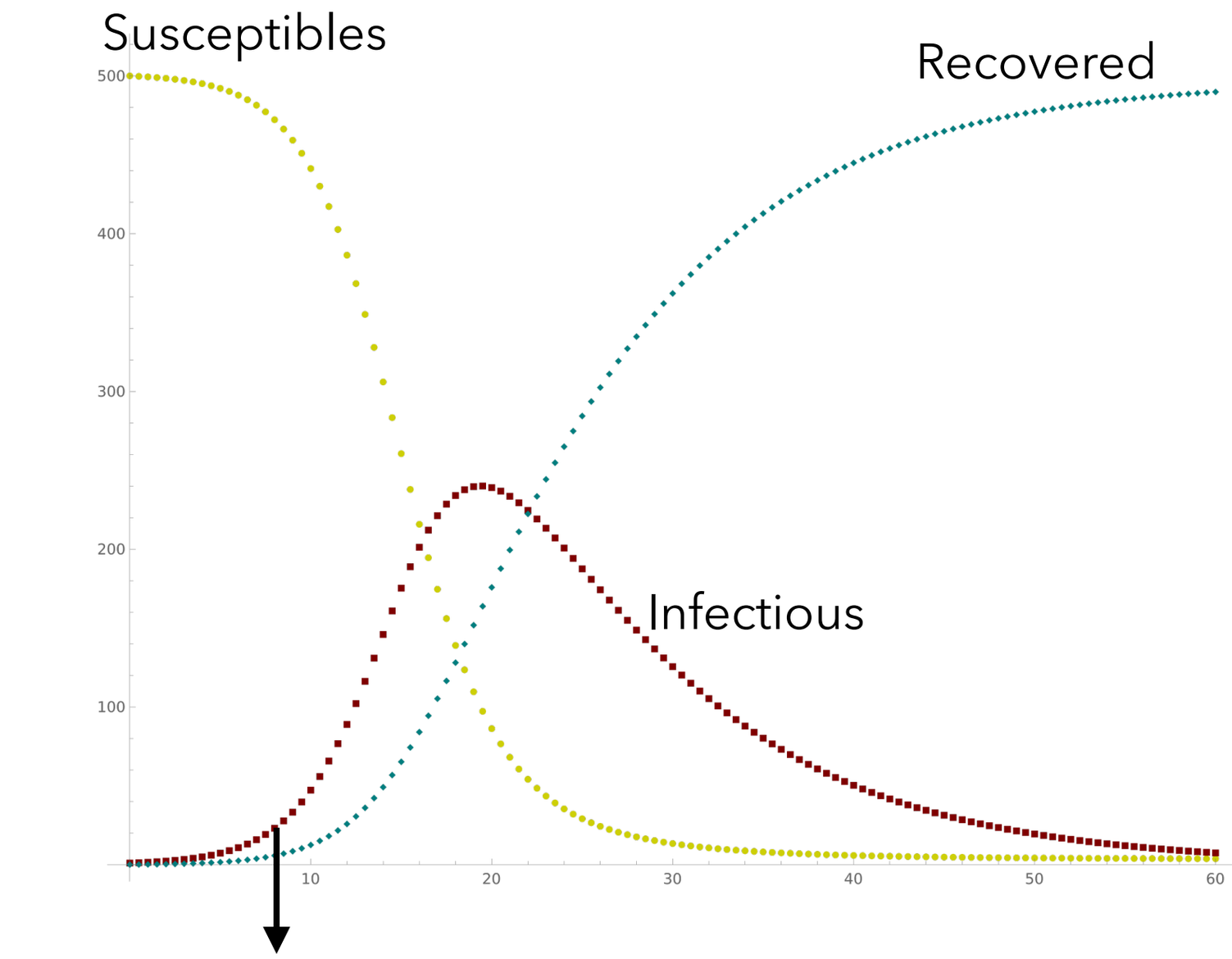
The epidemic terminates without reaching the whole population

Final attack rate = total fraction of infected at the end of the outbreak

This is function of R_0 only. It doesn't depend on β and μ separately

SIR Model

evolution of $s(t)$, $i(t)$ and $r(t)$ for $R_0 > 1$ and initial conditions, $s_0 \simeq 1$, $r_0 = 0$ and $i_0 \ll 1$



$$1 - r(\infty) = s_0 e^{-r(\infty)R_0}$$

$$i(\infty) \to 0$$

$$i(t) \simeq i_0 e^{(\beta\langle k\rangle - \mu)t} = i_0 e^{\mu(R_0 - 1)t}$$

SIR Model

Exponential growth $\beta\langle k\rangle - \mu = \mu(R_0 - 1)$

Final attack rate function of R_0 only

2 pathogens may have the same R_0, one being fast the other small

fast pathogen: high μ (short infectious period)

slow pathogen: small μ (short infectious period)