ON RATES, STATES, AND THE SIR MODEL

Modelling Biological Systems, BIOS13

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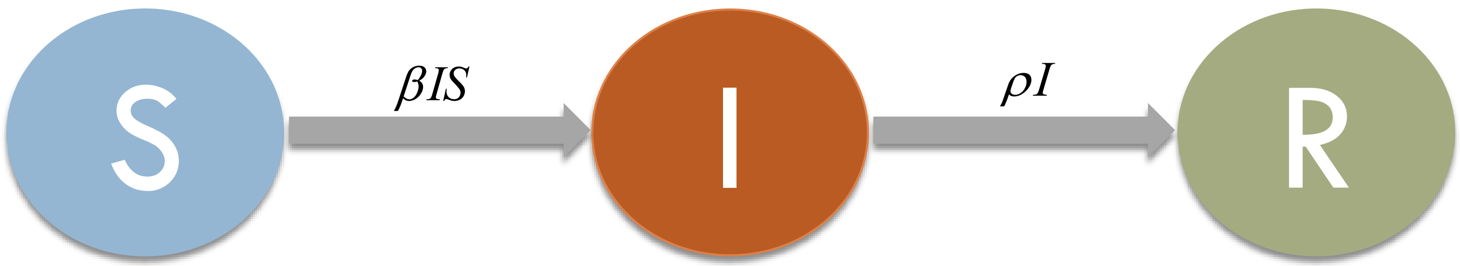
The SIR model

- The SIR model is a classic model from epidemiology (it actually is a class of models)
- The model describes the spread of a disease in a population
- It has three state variables: S, I and R
  - **S : Susceptibles** – the number of susceptible individuals
  - **I : Infected** – the number of infected individuals
  - **R : Recovered** – the number of recovered (immune) individuals

The basic SIR Model



$$\begin{cases} \dfrac{dS}{dt} = -\beta IS \\ \dfrac{dI}{dt} = \beta IS - \rho I \\ \dfrac{dR}{dt} = \rho I \end{cases}$$

S : Susceptibles
I : Infected
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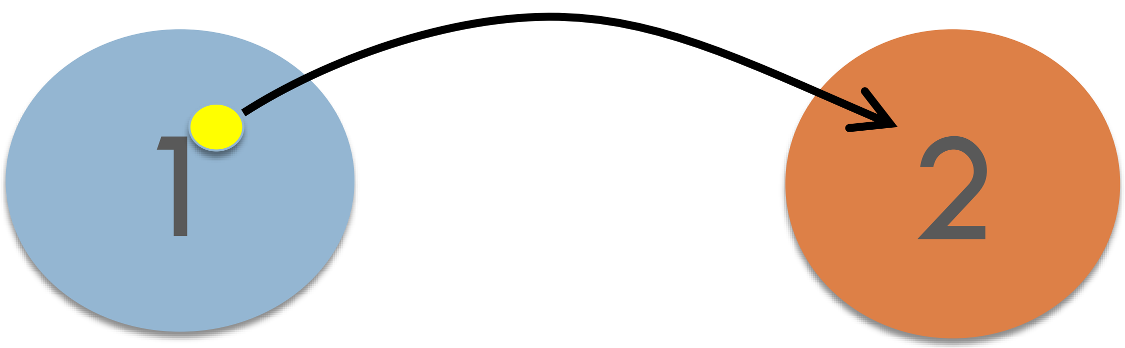
How SIR Models are Used

- SIR-based models were widely used early in the pandemic to estimate spread, peak timing, and healthcare needs.

- Fast early forecasts when little data existed
- Predicted exponential growth and peak infection
- Estimated hospital demand
- Informed lockdown and distancing strategies

Transition rates

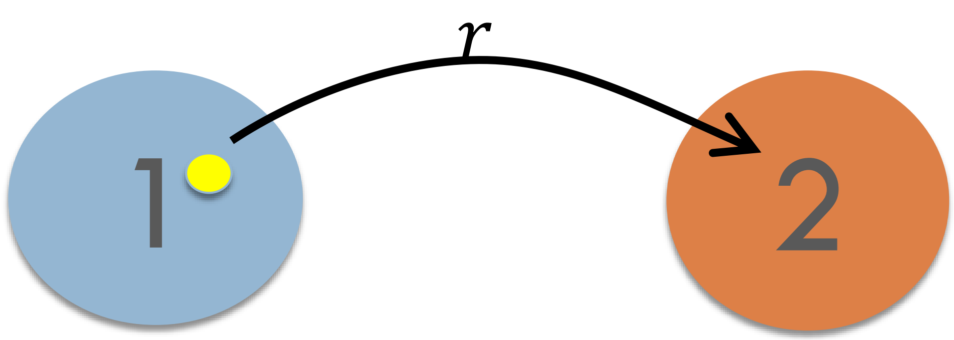
- A *rate* is a 'speed of change'. It it often a direct rate of change, such as a growth rate, dn/dt, but can also be a *transition rate*.
- *Transition rates* describe how frequently the transition from one state to another occurs.
  Examples: death rate (alive $\longrightarrow$ dead), dispersal rate (here $\longrightarrow$ there), or rate of a chemical reaction (compound 1 $\longrightarrow$ compound 2).



Transition rates, interpretation

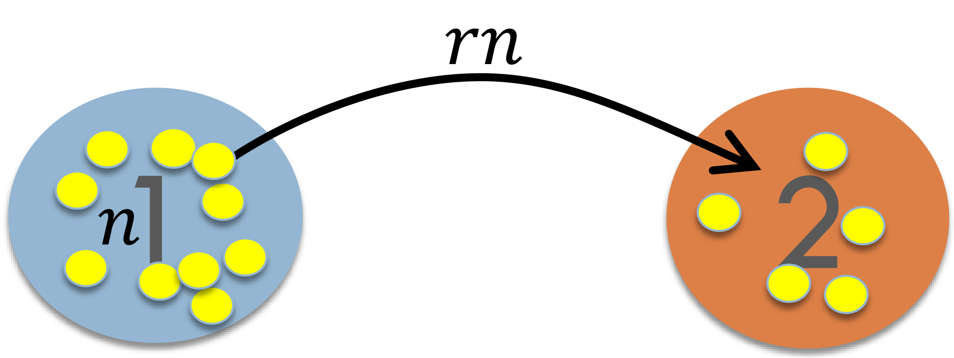
Transition probability

- Transition rates are (most often) probabilistic.
  The probability that a transition from state 1 to state 2 at rate r will occur in a time interval Δt is $r\Delta t$ (if Δt is small).
  Example: The probability that an individual with death rate 2/y (two per year) will die in a time interval $\Delta t = 0.01y$ is $2 \cdot 0.01 = 0.02$.
- An alternative, equivalent, interpretation: On average $r\Delta t$ transitions occur in the time interval Δt.
  Example: The individual above will die on average 0.02 times during that time interval.



Total transition rate

- Rates are often given *per item* (individual, or other object) or *per unit* (per mass unit, volume unit, or other).
  Examples: death rate per individual, decay rate per kg.
- The total rate at which a transition occurs is thus the per item rate multiplied by the number of items (e.g. individuals).
  Example: The total death rate of a population of size 1000 with a *per capita* death rate of 2/y is 2000/y. The average number of deaths in a time interval of 0.01y is thus 2000·0.01 = 20. (the probability interpretation fails here)



Application to COVID-19 Predictions

Transition rate × time interval allows estimation of:

- New infections per day
- Recoveries per day
- Expected hospitalization counts
- Expected mortality

- This informed ICU capacity planning and outbreak forecasting.

Average dynamics

- Even though the outcome is stochastic, it can be approximated by its average for large populations.
  For example, the group of infected in an SIR model decrease at a rate $-\rho I$.

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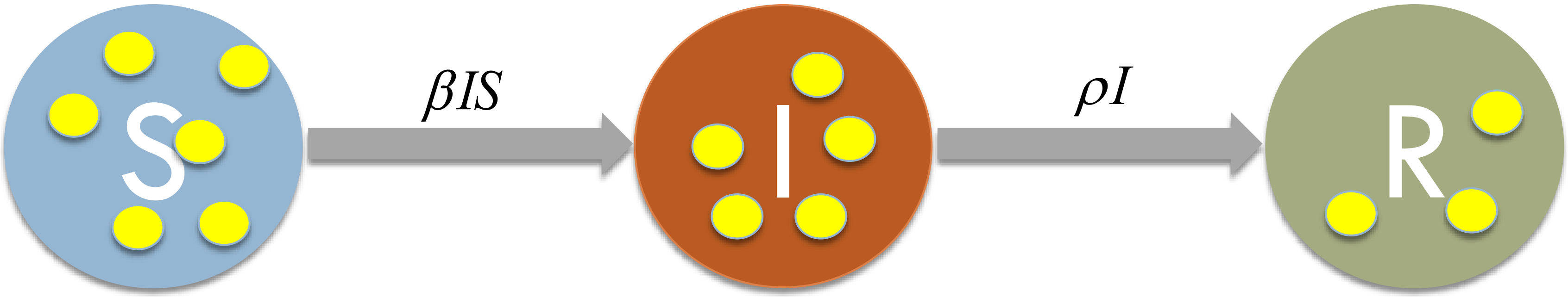
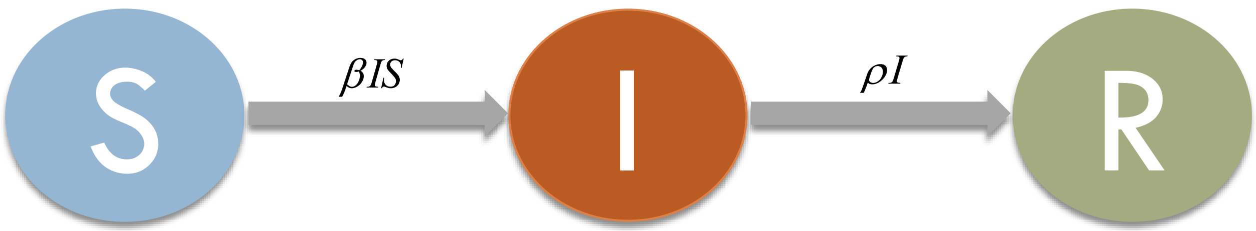


Average time to transition and R_0

- The average time an individual (or mass unit, or whatever) will stay in a state is $1/r$ if *r* is the transition rate *from* that state.
Example: The SIR model



$$\begin{cases} \dfrac{dS}{dt} = -\beta IS \\ \dfrac{dI}{dt} = \beta IS - \rho I \\ \dfrac{dR}{dt} = \rho I \end{cases}$$

The average time to recovery is $1/\rho$, i.e. the average time in illness. The higher the recovery rate, the shorter is the time an individual remains infected.
Additionally, each infected individual infects βS new individuals per time unit (the total infection rate is βIS).
The total number of new infected per infected individual is then $\frac{1}{\rho} \cdot \beta S = R_0$.
R_0 is a central concept in epidemiology. A disease will not spread as long as $\mathrm{R}_0 < 1$.

Applying SIR-Type Models to COVID-19

- If key epidemiological parameters are estimated from data, COVID-19 spread can be predicted.

Parameters typically estimated:

- β (infection/contact rate): estimated from case growth curves.
- ρ (recovery rate): estimated from duration of infectious period ($\approx 1/\rho$).
- Incubation period
- Initial number of infected individuals
- Population susceptibility at time of outbreak.

How it applies:

- Using β and ρ, we estimate $R_0 = \beta/\rho$.
- If $R_0 > 1 \rightarrow$ COVID-19 spreads; if $R_0 < 1 \rightarrow$ it decays.
- Predictions include expected peak infection, timing, and healthcare demand.

Some Covid-19 data

- R_0 has been estimated to between1.5 and 3.5
- Incubation period (from exposure to symptoms): 1-14 days (average 5.2)
- Recovery in 2 weeks (mild cases)

$$R_0 = \frac{1}{\rho} \cdot \beta S \qquad => \qquad \rho R_0 / S = \beta$$

Sources:
https://www.worldometers.info/coronavirus/
https://www.ecdc.europa.eu/en/covid-19/facts/questions-answers-basic-facts
https://ourworldindata.org/coronavirus

Conclusions from the COVID-19 Estimates

- Using early COVID-19 estimates ($R_0 \approx 1.5$–3.5 and ~14 days recovery):

- With an infectious duration of ~14 days $\rightarrow \rho \approx 1/14 \approx 0.07/\text{day}$.
- If $R_0 = 3$, then $\beta = \rho R_0 \approx 0.07 \times 3 \approx 0.21/\text{day}$.
- $\Rightarrow$ Each infected person infects ~0.21 new individuals per day.
- Over ~14 days $\rightarrow$ ~3 new infections per infected person.

- Since $R_0 > 1$, spread increases without interventions.
- Herd immunity threshold: $H = 1 - 1/R_0$
- For $R_0 = 3 \rightarrow H \approx 1 - 1/3 = 0.67 \rightarrow 67\%$ immunity needed to slow spread.

- Conclusion: With realistic parameter estimates, models could predict infection growth and inform vaccination and control measures.

Herd Immunity in COVID-19

Definition:

- Herd immunity occurs when enough individuals are immune so that the disease cannot spread easily through the population.

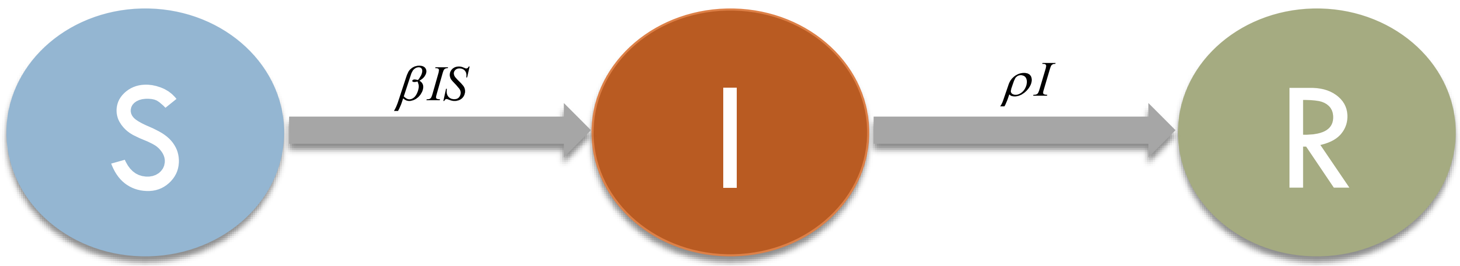
How it is calculated:

- $H = 1 - (1/R_0)$
- Example:
- If $R_0 = 3 \rightarrow H = 1 - 1/3 = 0.67 \rightarrow 67\%$ immunity required.

Why it matters:

- When immunity exceeds H, sustained outbreaks become unlikely.
- Determines vaccination coverage targets.
- Helps predict when transmission will decline naturally or due to interventions.

The basic SIR Model



$$
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$$

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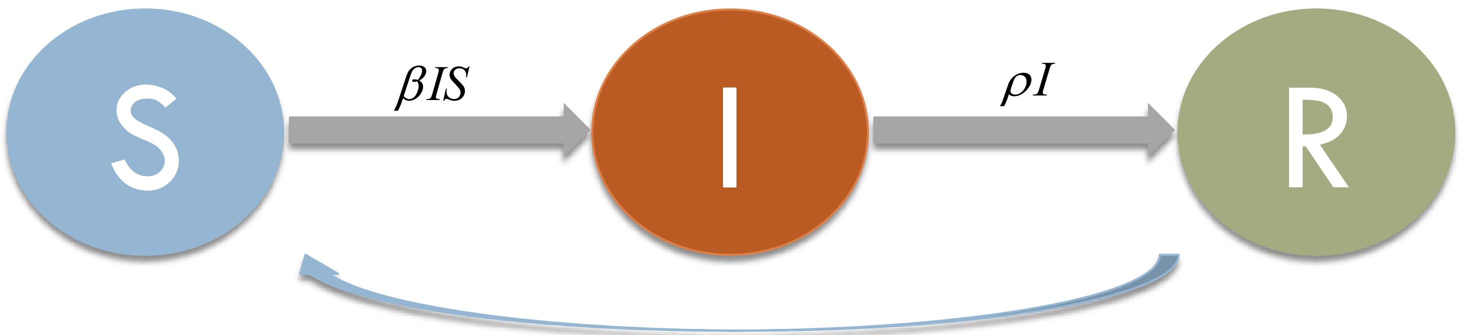
The basic SIR Model (limited resistance)



$$\frac{ds}{dt} = \beta IS + \gamma R$$

$$\frac{dI}{dt} = \beta IS - \rho I$$

$$\frac{dR}{dt} = \rho I - \gamma R$$

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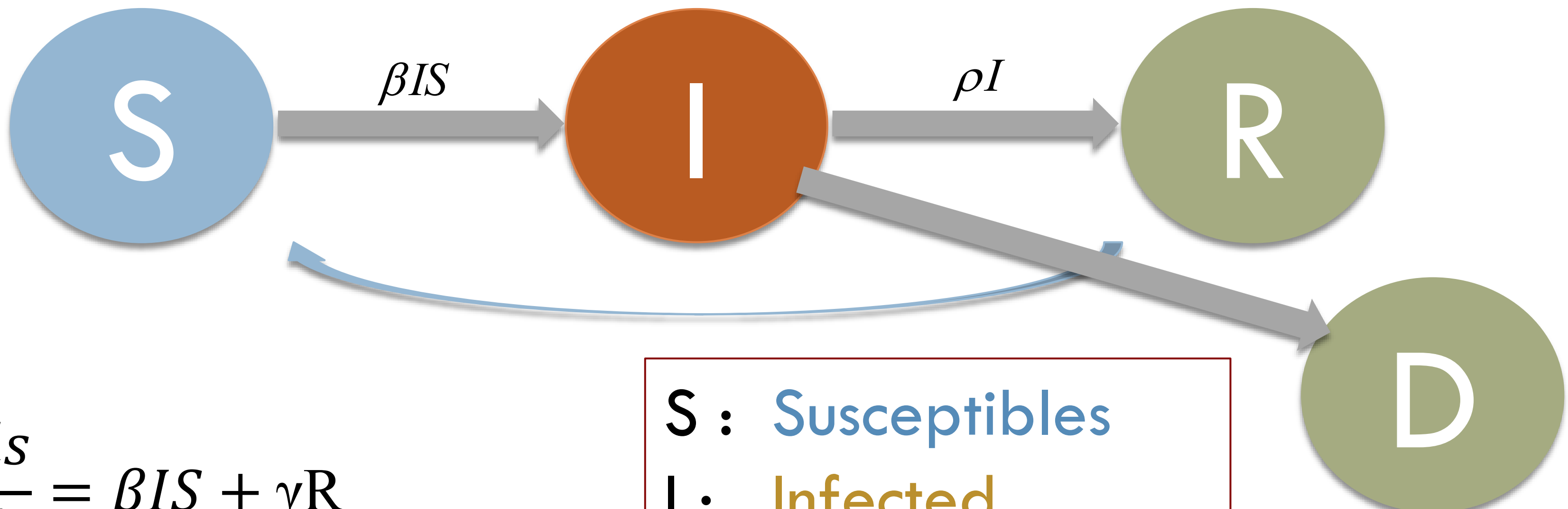
The basic SIR Model (death)



$$\frac{ds}{dt} = \beta IS + \gamma R$$

$$\frac{dI}{dt} = \beta IS - \rho I - \mu I$$

$$\frac{dR}{dt} = \rho I - \gamma R$$

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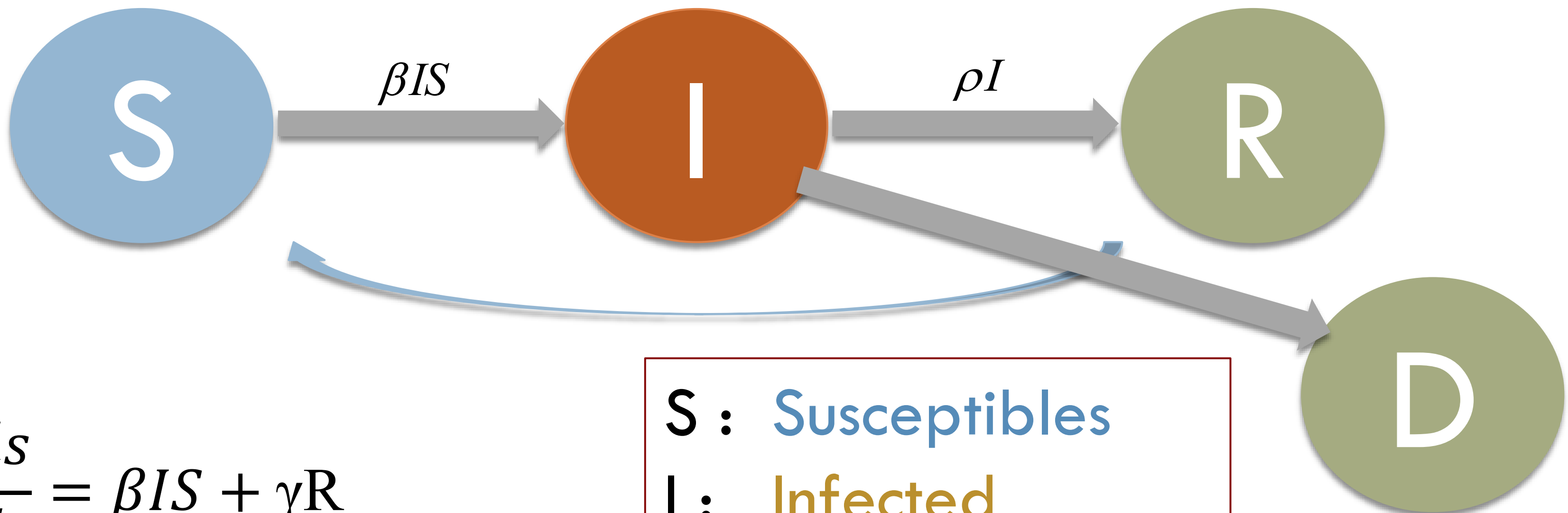
The basic SIR Model (death)



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… human birth, non-infected deaths, ect, ect

How Death Can Induce Cycles in Epidemic Dynamics

Death of infected individuals can reduce population size, temporarily lowering transmission and allowing susceptibles to recover or replenish, which may later trigger new outbreaks. This feedback can generate cyclic epidemic behaviour.

Why cycles arise:

- High mortality reduces susceptibles and transmission.
- As population slowly regenerates (births or recruitment), susceptible numbers rise.
- Once enough susceptibles exist, infection can spread again → new wave.

Other conditions that also generate cycles:

- • Waning immunity (R → S transitions).
- • Seasonal changes in transmission rates (e.g., $\beta(t)$ varies).
- • Demographic turnover (births replenish susceptible pool).
- • Behavioural changes over time (contact rates shift).

Why COVID-19 Showed Cyclic Waves

COVID-19 exhibited repeated waves, but not due to high mortality.

Key points:

- Death rate was too low to reduce population size enough to cause cycles.
- Waning immunity contributed, but was not the dominant driver.

Main causes of cycles:

- Seasonal changes in transmission (more indoor contact in winter).
- Behavioral shifts (restrictions, reopening, reduced compliance).
- Emergence of new variants (Alpha, Delta, Omicron).

Conclusion:

- COVID-19 waves arose from changes in transmission conditions over time,
- not from high death-induced demographic effects.

Herd Immunity and Vaccination Programs

Herd immunity occurs when a sufficient fraction of the population is immune, preventing sustained transmission.

How it is calculated:

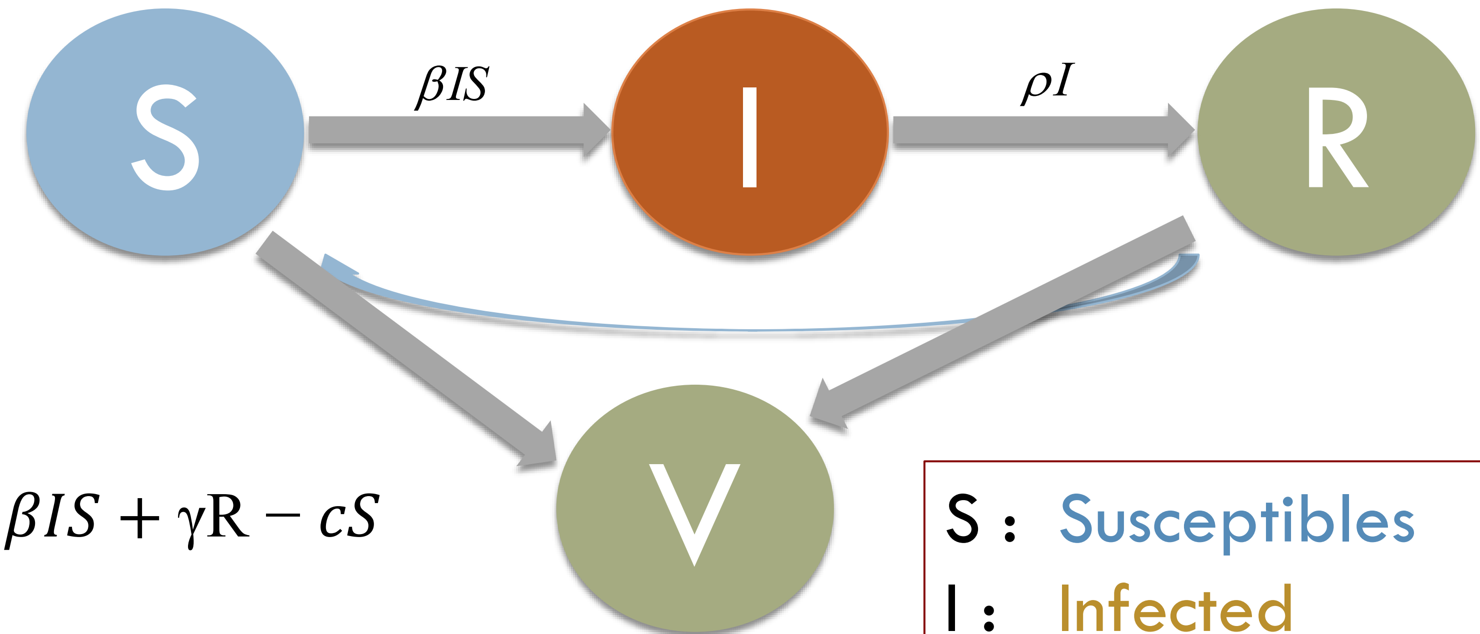
- $H = 1 - 1/R_0$
- Example: If $R_0 = 3 \rightarrow H \approx 67\%$ immunity required.

Connection to vaccination programs:

- Vaccination increases immune individuals without requiring infection.
- When coverage exceeds H, outbreaks decline naturally.

- Conclusion: Vaccination programs aim to raise immunity above the herd immunity threshold, reducing long-term disease spread.

The basic SIR Model (vaccination)



$$\frac{ds}{dt} = \beta IS + \gamma R - cS$$

$$\frac{dI}{dt} = \beta IS - \rho I$$

$$\frac{dR}{dt} = \rho I + \gamma R - cR$$

$$\frac{dV}{dt} = c(S + R)$$

S : Susceptibles

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Permanent Vaccination and Disease Elimination

If vaccination leads to permanent immunity and coverage exceeds the herd immunity threshold, infection will eventually disappear.

Reason:

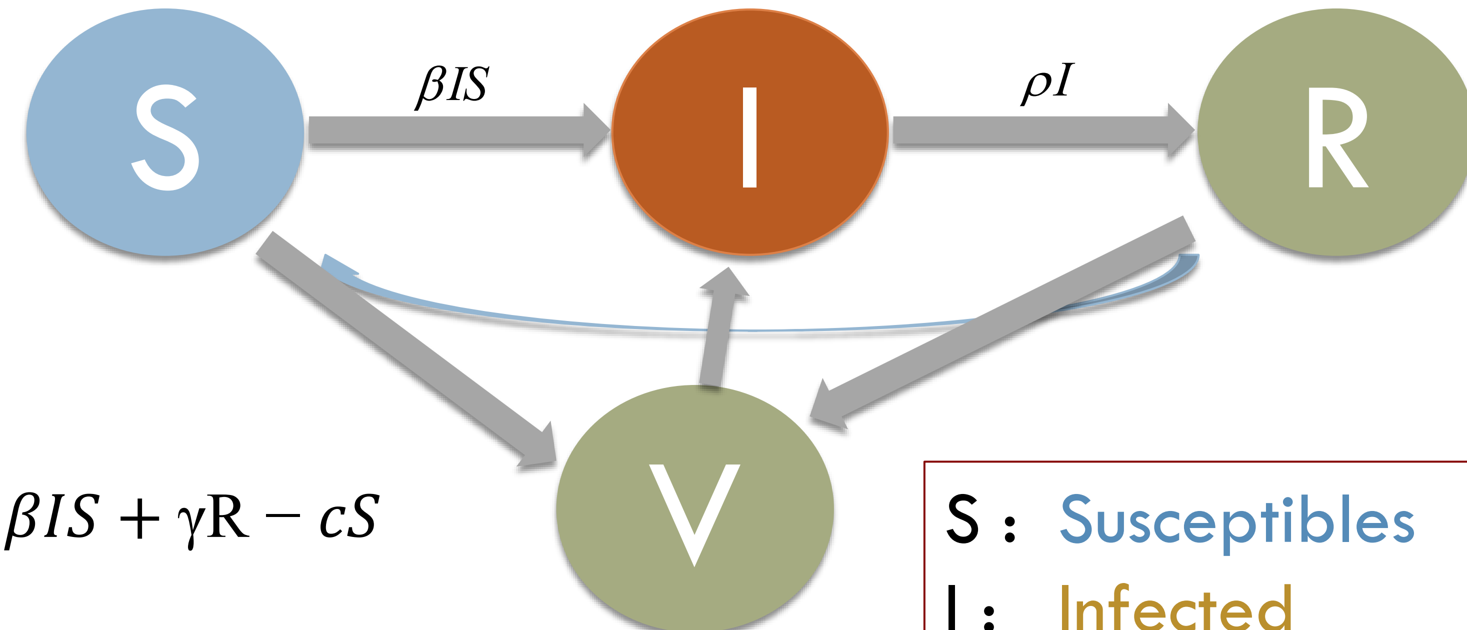
- Susceptible pool shrinks permanently.
- Effective reproduction number falls below 1:

$$R\_eff = R_0 \times S < 1.$$

Outcome:

- Infection declines exponentially and dies out.
- Same mechanism enabled smallpox eradication.

The basic SIR Model (vaccination)



$$\frac{ds}{dt} = \beta IS + \gamma \mathrm{R} - cS$$

$$\frac{dI}{dt} = \beta IS - \rho \mathrm{I} + e\beta I\mathrm{V}$$

$$\frac{dR}{dt} = \rho \mathrm{I} + \gamma \mathrm{R}\text{-}\mathrm{cR}$$

$$\frac{dV}{dt} = c(S + R) - e\beta I\mathrm{V}$$

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Imperfect Vaccination and Long-Term Coexistence

If vaccination is imperfect, infection persists and becomes endemic.

- Effective immunity fraction = $p \times v$
- where p = vaccination coverage and v = vaccine efficacy.

- If $p \times v < 1 - 1/R_0$, herd immunity is not reached → infection remains.

Why coexistence occurs:
- Some vaccinated individuals still transmit.
- Immunity may wane.
- Reinfections become possible.

Examples: Influenza, COVID-19, etc, etc.
- Result: Stable endemic disease rather than eradication.