From Equations to Impact

The Art of Modeling Infectious Diseases

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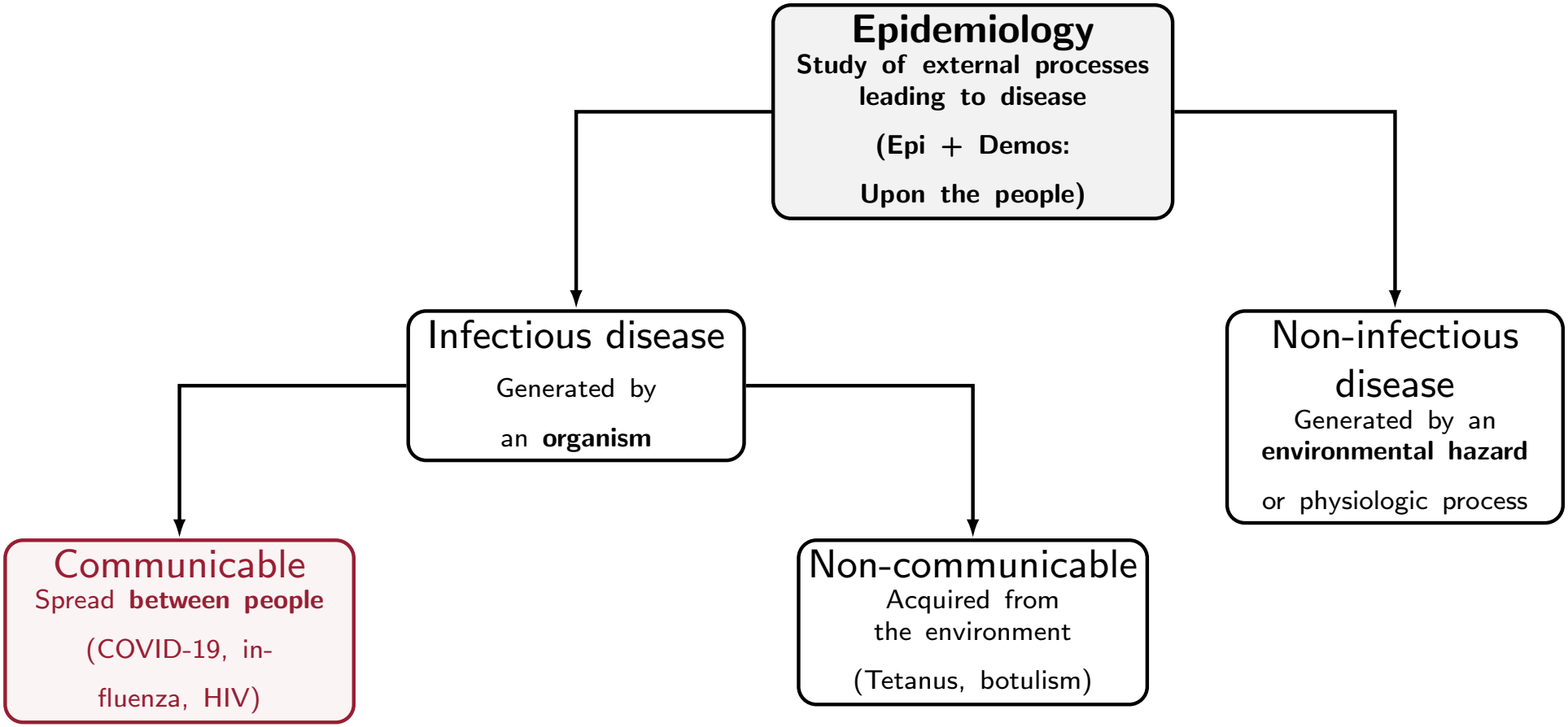
Mathematical Epidemiology — Data Analytics — Public Health Modeling

Outline

Epidemiology: Classification of Diseases

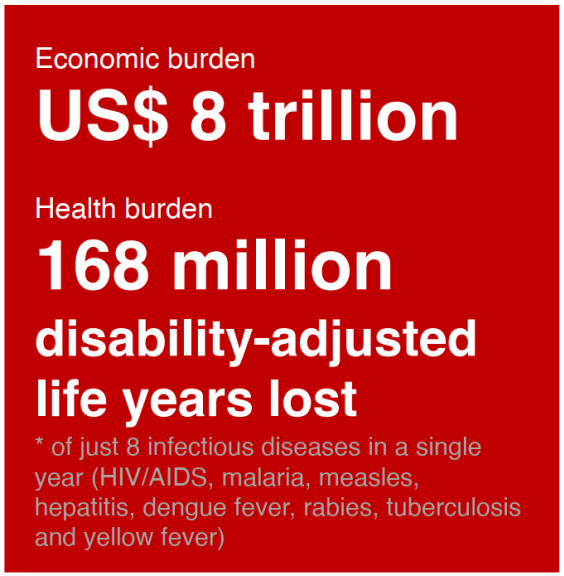


What are Infectious Diseases?

- Many agents infect humans, animals, and plants
- Transmission: direct (droplets, fluids) or indirect (vectors, environment)
- Microparasites: viruses, bacteria, fungi
- Macroparasites: worms
- Pathogens = infectious agents causing disease

Global Burden of Infectious Diseases

- **Economic burden:** estimated at nearly $8 trillion per year for only eight major infectious diseases (HIV/AIDS, malaria, measles, hepatitis, dengue, rabies, tuberculosis, yellow fever).
- **Health burden:** about 168 million disability-adjusted life years (DALYs) lost.
- For perspective: U.S. billionaires' combined net worth $4.5 trillion; Germany's GDP $4.1 trillion.



Characteristics of Infectious Diseases

- Transmission route & potential
- Latent (pre-infectious) and infectious period
- Incubation time
- Acquired immunity
- Symptomatic vs asymptomatic cases

Transmission Routes of Infectious Diseases

- **Respiratory:** Spread through droplets or aerosols released when an infected person coughs, sneezes, or breathes. *Examples:* Measles, influenza, COVID-19.
- **Sexual:** Transmitted through sexual contact via semen, vaginal fluids, or mucosal surfaces. *Examples:* HIV, HPV, gonorrhea.
- **Orofecal (Fecal-oral):** Pathogens in fecal matter contaminate food or water, which is then ingested. *Examples:* Cholera, hepatitis A, typhoid fever.
- **Parenteral (Blood-borne):** Direct entry into the bloodstream through needles, transfusions, or open wounds. *Examples:* Hepatitis C, HIV.
- **Vertical:** Transmission from mother to child during pregnancy, birth, or breastfeeding. *Examples:* HIV, hepatitis B, rubella.
- **Vector-borne:** Spread by insects or other arthropods that carry pathogens between hosts. *Examples:* Malaria, dengue, Zika virus.

Vector-Borne Transmission Examples

Malaria (Anopheles mosquito)

- Active between sunset and sunrise.
- Breeds in natural water bodies.
- Multiple hosts.

Dengue (Aedes mosquito)

- Daytime feeders, highly domesticated.
- Humans are preferred hosts.
- Prevention: removal of open water containers.

Anopheles

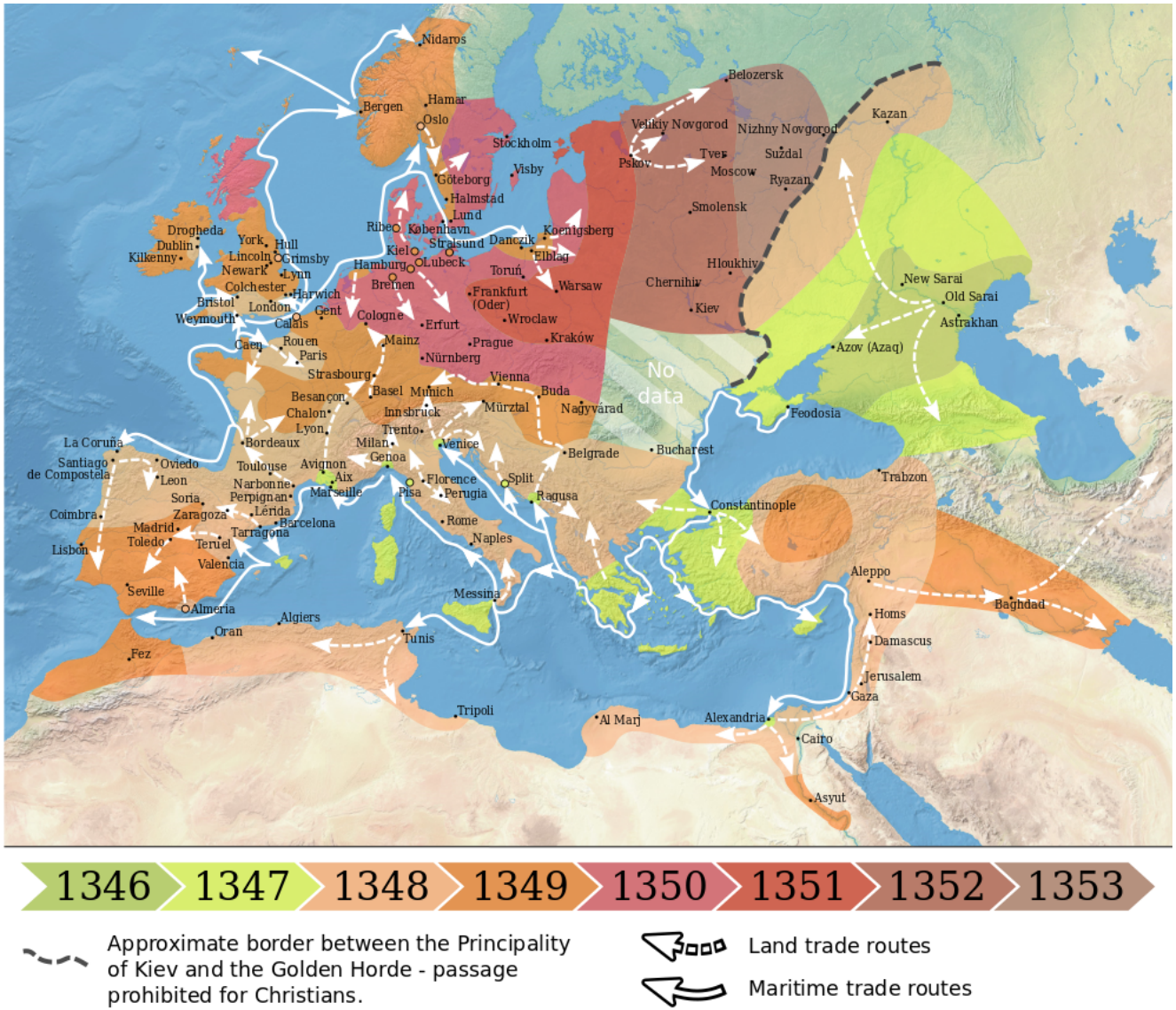
Image: Wikipedia

Aedes

Historic Pandemics

- Plague of Justinian (541–750): killed 50–60% of Europe's population.
- Black Death (1347–1352): 25–50% mortality across Europe, Asia, and Africa.
- Smallpox and measles in the Americas (15th–16th centuries): massive population decline among Indigenous peoples.
- 1918 Influenza ("Spanish Flu"): 25–50 million deaths worldwide.
- HIV/AIDS (since 1980s): over 40 million deaths globally.
- **COVID-19 (2019–present):** Caused by the SARS-CoV-2 coronavirus, first identified in Wuhan, China, in late 2019. Declared a pandemic by WHO in March 2020, it led to major global disruption, with more than 770 million confirmed cases and nearly 7 million reported deaths by 2024 (WHO). Rapid vaccine development and public health interventions dramatically reduced mortality rates after 2021.

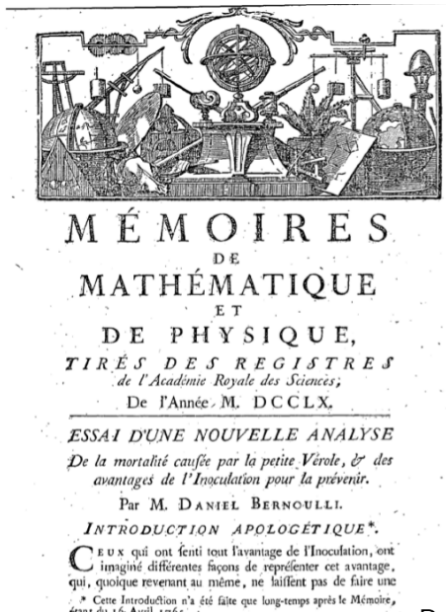
Spread of the Black Death (1346–1353)



Source: Wikimedia Commons, *"Spread of the Black Death in Europe (1346–1353)"*.

Where Did Infectious Disease Models Come From?

Daniel Bernoulli (1700–1782)



MÉMOIRES
DE
MATHÉMATIQUE
ET
DE PHYSIQUE,
TIRÉS DES REGISTRES de l'Académie Royale des Sciences;
De l'Année M. DCCLX.

ESSAI D'UNE NOUVELLE ANALYSE
De la mortalité causée par la petite Vérole, & des avantages de l'Inoculation pour la prévenir.
Par M. DANIEL BERNOULLI.
INTRODUCTION APOLOGÉTIQUE.*
CEUX qui ont senti tout l'avantage de l'Inoculation, ont imaginé différentes façons de représenter cet avantage, qui, quoique revenant au même, ne laissent pas de faire une

Essai d'une nouvelle analyse... (1766)

Bernoulli's Model of Smallpox Mortality

- Published in 1766 in the *Mémoires de Mathématique et de Physique*.
- One of the first mathematical models of infectious disease.
- Estimated the increase in life expectancy from smallpox inoculation.
- Used life tables and differential equations for infection and survival.
- Pioneered quantitative reasoning in public health.

From Bernoulli to the SIR Framework

The Evolution of Epidemic Modeling

1766 $\rightarrow$ 1915/1927 $\rightarrow$ Today

Bernoulli's life-table model $\rightarrow$ early differential epidemic theory $\rightarrow$ compartmental SIR/structural/agent-based models

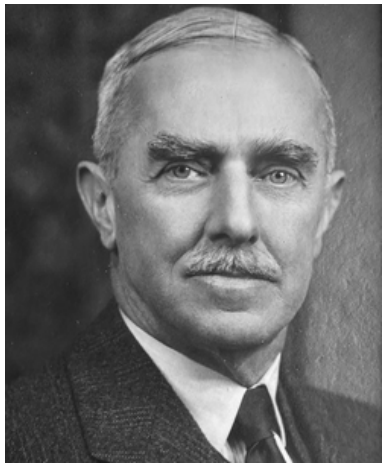
Key milestones:

- **1766 — Daniel Bernoulli:** first quantitative model of smallpox inoculation using life tables.
- **1915 — Early differential model:** first explicit use of equations for population infection dynamics, laying groundwork for the SIR model.
- **1927 — Kermack & McKendrick:** formal SIR framework describing epidemic growth, threshold, and final size.

1915 reference: early "Theory of Happenings" paper published in the *Proceedings of the Royal Society*, introducing differential infection rates for affected vs non-affected groups.

A Contribution to the Mathematical Theory of Epidemics

William O. Kermack (1898–1970)



Anderson G. McKendrick (1876–1943)

Landmark paper: W. O. Kermack A. G. McKendrick. "A Contribution to the Mathematical Theory of Epidemics." *P*roceedings of the Royal Society of London A, Vol 115 (772): 700-721, Aug 1 1927

- Introduced the concept of Susceptible → Infectious → Removed (S→I→R).
- Derived threshold conditions for an epidemic (implicit precursor to R_0).
- Provided analytical insights into epidemic growth, peak, and final size.
- Represented a foundational shift from life-tables to flow models of transmission.

Why Mathematical Models?

- **Understand disease dynamics:** Models describe how infections spread, persist, or die out in a population — identifying key drivers such as contact rate, immunity, and seasonality.
- **Predict future outbreaks:** By estimating parameters like R_0 or transmission rates, models help forecast epidemic peaks and total cases, guiding public health planning.
- **Evaluate interventions:** Quantitatively assess the impact of vaccination, quarantine, mask use, or vector control. *Example:* Estimating how high vaccination coverage must be to achieve herd immunity.
- **Explore "what-if" scenarios:** Models allow simulation of different assumptions — e.g., emergence of a new variant or changes in contact patterns — to support preparedness.

Trade-offs:

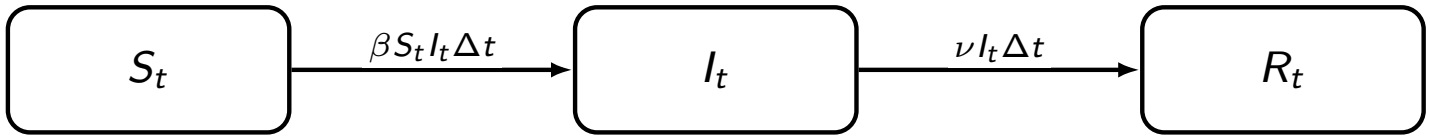
- **Accuracy:** More complex models may capture details better but need more data.

What Can't Mathematical Models Do?

- **Predict the future with certainty:** Models are not crystal balls — they depend on assumptions and input data, which can change as new variants, behaviors, or interventions emerge.
- **Capture every detail of reality:** Simplifications are necessary — models ignore many biological, environmental, and social complexities to remain tractable.
- **Compensate for poor or missing data:** Even the best model fails if parameters or surveillance data are unreliable or biased.
- **Replace expert judgment or policy decisions:** Models inform decisions, but public health actions also rely on ethics, economics, and social factors.
- **Eliminate uncertainty:** Every model output includes error; interpreting uncertainty is part of responsible modeling.

Key takeaway: *Models are simplified representations — useful for insight and planning, not perfect predictions.*

Discrete-Time SIR Model



$\Delta t = 1$ step: $S_{t+\Delta t} = S_t - \beta S_t I_t \Delta t$, $I_{t+\Delta t} = I_t + \beta S_t I_t \Delta t - \nu I_t \Delta t$, $R_{t+\Delta t} = R_t + \nu I_t \Delta t$

- Time advances in discrete steps (e.g., days or weeks).
- At each step, a fraction of susceptible individuals becomes infected.
- This approach aligns naturally with surveillance data reported per day or week.

$$
\begin{aligned}
S_{t+1} &= S_t - \beta S_t I_t, \\
I_{t+1} &= I_t + \beta S_t I_t - \nu I_t, \\
R_{t+1} &= R_t + \nu I_t.
\end{aligned}
$$

Each "bucket" loses or gains individuals at each step.

The Basic Reproduction Number R_0 (Discrete Intuition)

- At the start of an epidemic:

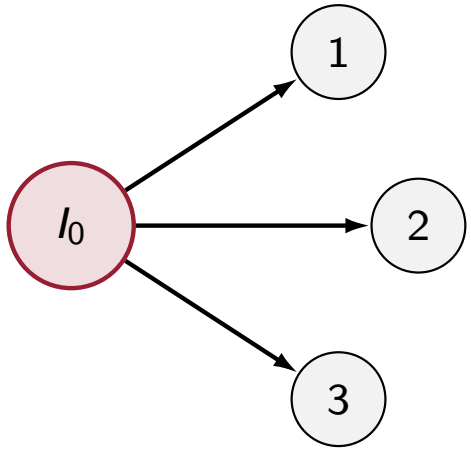
$$S_0 \approx N, \quad I_0 = 1.$$

- New infections per unit time: $\text{new}_t = \beta S_t I_t \approx \beta N$.
- Over an infectious period D:

$$R_0 = \beta N D.$$

- **Interpretation:** average number of secondary infections caused by a single infective in a fully susceptible population.

Assumes homogeneous mixing, constant β and D.



Each infected person generates R_0 new infections on average.

Interpreting R_0 as a Threshold

- R_0 is a **threshold parameter:**

$$\begin{cases} R_0 > 1 & \text{epidemic can grow,} \\ R_0 = 1 & \text{steady state,} \\ R_0 < 1 & \text{epidemic will die out.} \end{cases}$$

- Using R_0, we define the number of **effective contacts per person per unit time**:

$$c_e = \frac{R_0}{D}.$$

- The **per capita contact rate** (rate at which a given individual makes effective contact per unit time):

$$\beta = \frac{c_e}{N} = \frac{R_0}{ND}.$$

- **Note:** For a given pathogen, defining an "effective contact" is not always simple — it depends on mode of transmission, environment,

From Discrete to Continuous Models

- Let Δt be a small time step.
- The difference quotient $\dfrac{S_{t+\Delta t} - S_t}{\Delta t}$ becomes a derivative as $\Delta t \to 0$.

$$\frac{dS}{dt} = -\beta SI, \qquad \frac{dI}{dt} = \beta SI - \nu I, \qquad \frac{dR}{dt} = \nu I.$$

Interpretation: The continuous model captures instantaneous change, while the discrete model follows case counts step by step.

Scaling the SIR Model by Population Size

- In the continuous SIR model, we used absolute counts:

$$\frac{dS}{dt} = -\beta SI, \qquad \frac{dI}{dt} = \beta SI - \nu I, \qquad \frac{dR}{dt} = \nu I.$$

- Let total population be $N = S + I + R$ (constant for a closed system).
- Define **normalized variables:**

$$s = \frac{S}{N}, \quad i = \frac{I}{N}, \quad r = \frac{R}{N}.$$

  Then $s + i + r = 1$.
- Substituting into the system gives:

$$\frac{ds}{dt} = -\beta si, \qquad \frac{di}{dt} = \beta si - \nu i, \qquad \frac{dr}{dt} = \nu i.$$

  Now, the equations depend only on β and ν, not on N.
- **Interpretation:** Scaling by N expresses the model in terms of **fractions of the population**, making it dimensionless and easier to compare across populations of different sizes.

The Basic Reproduction Number R_0 in the Continuous SIR Model (Scaled Form)

Normalized (per capita) SIR model:

$$\frac{ds}{dt} = -\beta si, \qquad \frac{di}{dt} = \beta si - \nu i, \qquad \frac{dr}{dt} = \nu i, \qquad s + i + r = 1.$$

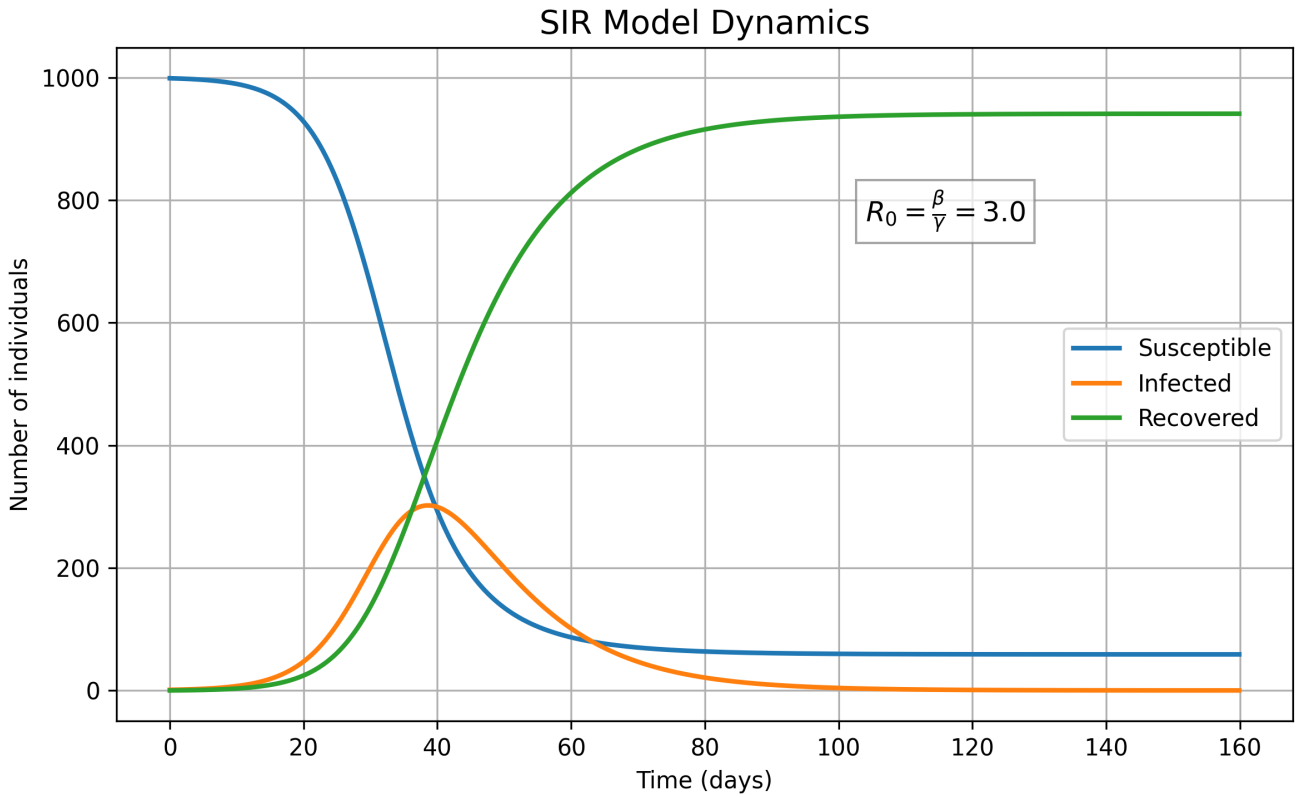
- Here, $s = S/N$, $i = I/N$, and $r = R/N$ are **fractions** of the population.
- β is the **effective contact rate** (per person per unit time).
- ν is the **recovery rate**.
- At the start of an epidemic, $s(0) \approx 1$, so:

$$\frac{di}{dt} \approx (\beta - \nu)\, i.$$

- Infection grows if $\beta > \nu$.
- Therefore, the **basic reproduction number** is:

$$\boxed{R_0 = \frac{\beta}{\nu}}.$$

Results of the SIR Model Simulation



Key observations:

- The epidemic curve shows a rapid increase in infections followed by decline as susceptibles are depleted.
- $R_0 = \beta/\gamma$ controls whether the epidemic grows ($R_0 > 1$) or fades ($R_0 < 1$).
- Final epidemic size depends on both initial conditions and R_0.
  Example parameters: $\beta = 0.3$, $\gamma = 0.1$, $R_0 = 3$, initial $I(0) = 1$, $S(0) = 999$.

Limitations of the Basic SIR Model

- The SIR model provides a **simplified framework** to understand epidemic spread.
- It captures the **essential mechanism:** infection and recovery transitions.
- However, it makes several simplifying assumptions:
  - **Closed population:** no births, deaths, or migration.
  - **Homogeneous mixing:** every individual has the same probability of contact.
  - **Constant parameters:** transmission rate (β) and recovery rate (ν) do not change over time.
  - **No interventions:** no vaccination, quarantine, or behavioral changes.
  - **No latent or exposed period:** infection occurs immediately after contact.
- **Consequence:** The model is useful for conceptual understanding and basic analytics, but less realistic for policy analysis or long-term forecasts.

Next steps: Relax these assumptions $\rightarrow$ include vital dynamics, incubation, and

SIR Model with Vital Dynamics

- We now include **births and deaths** at rate μ:
  - Births introduce new susceptibles into the population.
  - Deaths remove individuals from all compartments equally.

Model (fractions of total population):

$$\frac{ds}{dt} = \mu - \beta si - \mu s,$$

$$\frac{di}{dt} = \beta si - (\nu + \mu) i,$$

$$\frac{dr}{dt} = \nu i - \mu r, \qquad s + i + r = 1.$$

Equilibria:

$$E_0 = (1, 0, 0), \qquad E_1 = \left(\frac{\mu+\nu}{\beta}, \frac{\mu}{\beta}\left(\frac{\beta}{\mu+\nu} - 1\right), 1 - s_1 - i_1 \right).$$

Threshold condition: $R_0 = \frac{\beta}{\mu+\nu}$.

If $R_0 > 1$, the infection becomes **endemic***; if $R_0 < 1$, it dies out.*

Interpreting R_0 with Demography

- **Transmission rate:** β — average number of effective contacts per person per unit time.
- **Recovery rate:** $\nu = 1/D$ — average duration of infectiousness (D days).
- **Birth/death rate:** $\mu = 1/L$ — average lifespan (L time units).
- The **expected infectious period including mortality:**

$$\frac{1}{\mu + \nu}.$$

- Hence,

$$\boxed{R_0 = \frac{\beta}{\mu + \nu}}$$

  is the average number of secondary infections produced by one infectious individual in a demographically open population.
- If $R_0 > 1$, the infection persists at a positive equilibrium (endemic state); if $R_0 < 1$, it fades out.

Vaccination at Birth and Herd Immunity

- Suppose a fraction p of newborns is **vaccinated** at birth.
- The vaccinated enter the R (immune) compartment immediately:

$$\frac{ds}{dt} = \mu(1-p) - \beta si - \mu s,$$

$$\frac{di}{dt} = \beta si - (\nu + \mu) i,$$

$$\frac{dr}{dt} = \mu p + \nu i - \mu r.$$

- **Equilibria:**

$$E_0 = (1-p,\, 0,\, p), \qquad \text{endemic if } (1-p)R_0 > 1.$$

Critical vaccination coverage:

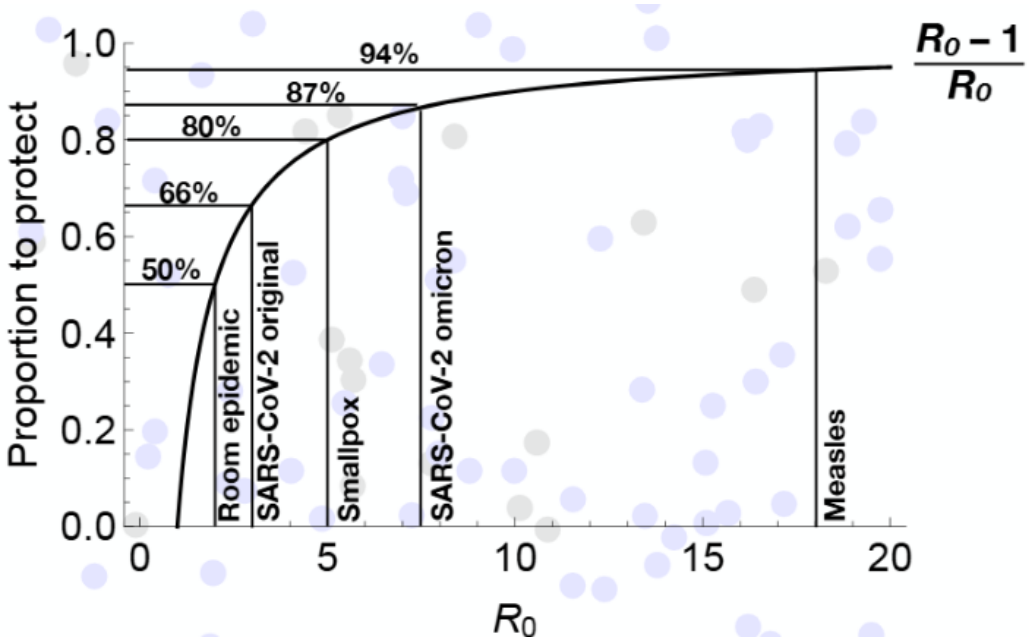
$$p_c = 1 - \frac{1}{R_0}.$$

Effective reproduction number:

$$R_e = (1-p)R_0.$$

Herd immunity occurs when $R_e < 1$ or equivalently $p > p_c$.

Herd Immunity Threshold



Concept:

- When a sufficient fraction of the population is immune, disease transmission cannot sustain.
- The **critical vaccination fraction**:

$$p_c = \frac{R_0 - 1}{R_0}$$

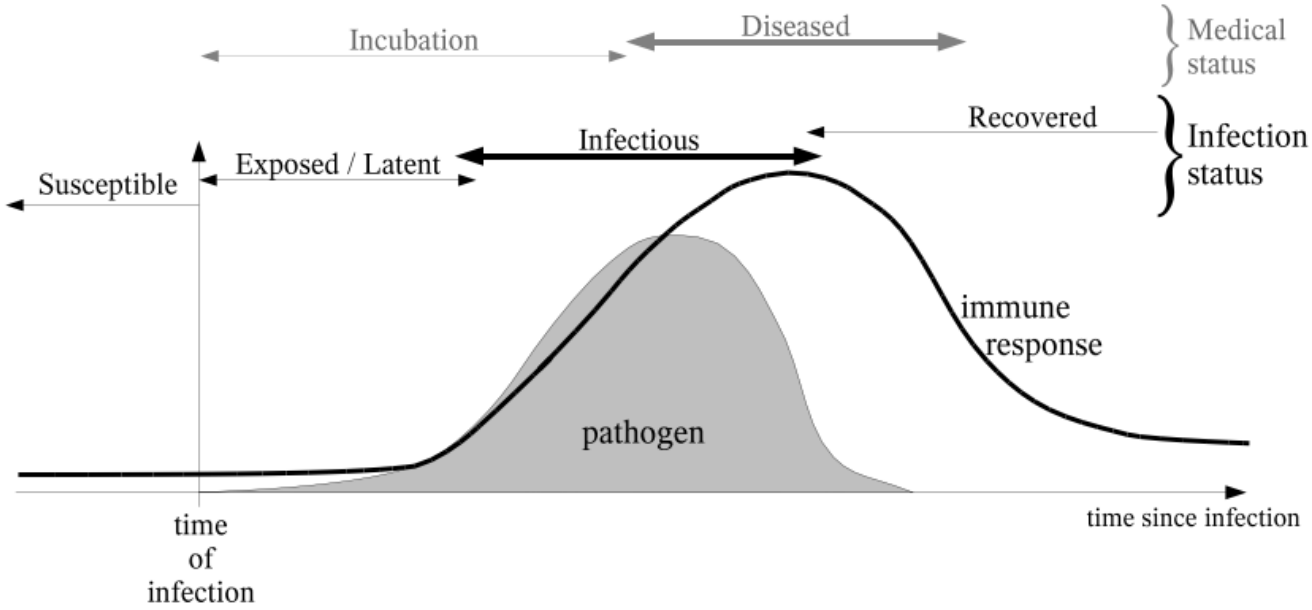
- Example: for $R_0 = 3$, about 67% must be immune to stop spread.

Implication:

- Herd immunity protects those who cannot be vaccinated.

Natural History of Infection

The Infectious Disease Process:



Adapted from: Keeling and Rohani (2008), *Modeling Infectious Diseases in Humans and Animals*.
Illustrates biological and model-based stages: Susceptible → Exposed (latent) → Infectious → Recovered.

The SEIR Model

- The SEIR model extends the SIR framework by adding an **Exposed (E)** compartment.
- Individuals pass through four stages:

$$S \xrightarrow{\text{infection}} E \xrightarrow{\text{latent period}} I \xrightarrow{\text{recovery}} R$$

- **Interpretation:**
  - S: Susceptible — can acquire infection.
  - E: Exposed — infected but not yet infectious (latent phase).
  - I: Infectious — capable of transmitting the pathogen.
  - R: Recovered/Removed — immune or isolated.

The SEIR Model

Model equations:

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

$$\frac{dE}{dt} = \beta SI - \sigma E,$$

$$\frac{dI}{dt} = \sigma E - \nu I,$$

$$\frac{dR}{dt} = \nu I.$$

Parameters: β — transmission rate, $\sigma = 1/L$ — rate of leaving latent period (L: mean latency), $\nu = 1/D$ — recovery rate (D: infectious duration).

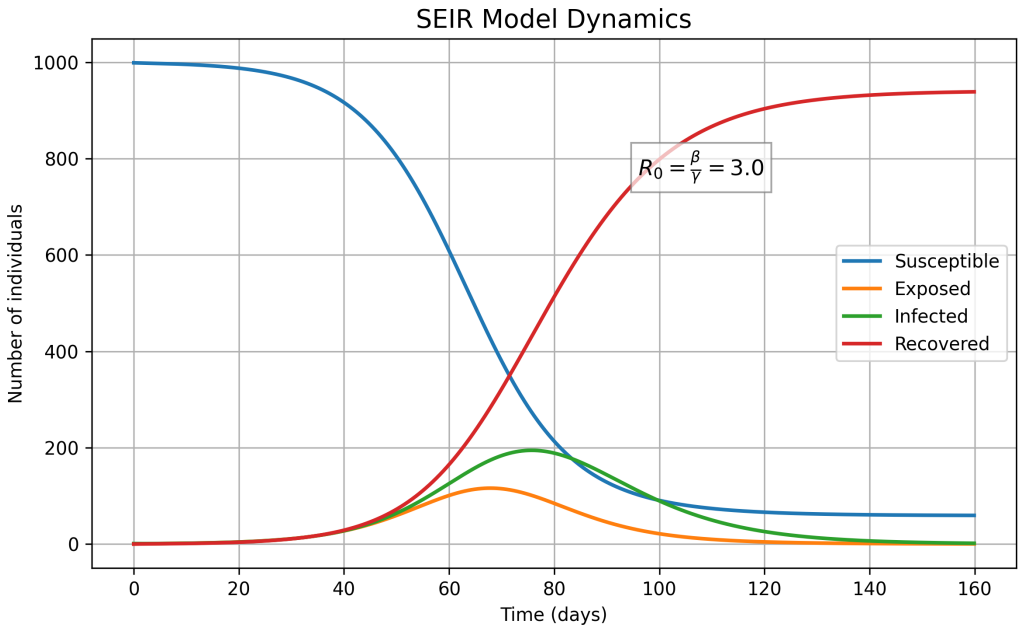
SIR vs SEIR: Why Add an Exposed Class?

SIR Model

- Individuals become infectious *immediately* after infection.
- Good approximation for diseases with negligible latent period.
- Peaks earlier and higher.

SEIR Model

- Adds a **latent (Exposed) compartment** E.
- Captures incubation: infected but not yet infectious.
- Peak infections occur later and are reduced.



SEIR dynamics: latency delays and lowers the epidemic peak.

MSEIR Model: Adding Maternal Immunity

Motivation:

- Some diseases (e.g., measles, rubella) involve **maternal immunity**: newborns receive antibodies from their mothers.
- These infants are **temporarily protected** and enter a new compartment M.
- When maternal antibodies wane (at rate γ), they move into the susceptible class.

Model equations:

$$\begin{aligned}
\frac{dM}{dt} &= \mu N - (\gamma + \mu)M, \\
\frac{dS}{dt} &= \gamma M - (\lambda + \mu)S, \\
\frac{dE}{dt} &= \lambda S - (\kappa + \mu)E, \\
\frac{dI}{dt} &= \kappa E - (\nu + \mu)I, \\
\frac{dR}{dt} &= \nu I - \mu R, \\
\lambda &= \beta \frac{I}{N}.
\end{aligned}$$

Basic reproduction number:

$$\boxed{R_0 = \frac{\kappa\,\beta}{(\kappa + \mu)(\nu + \mu)}.}$$

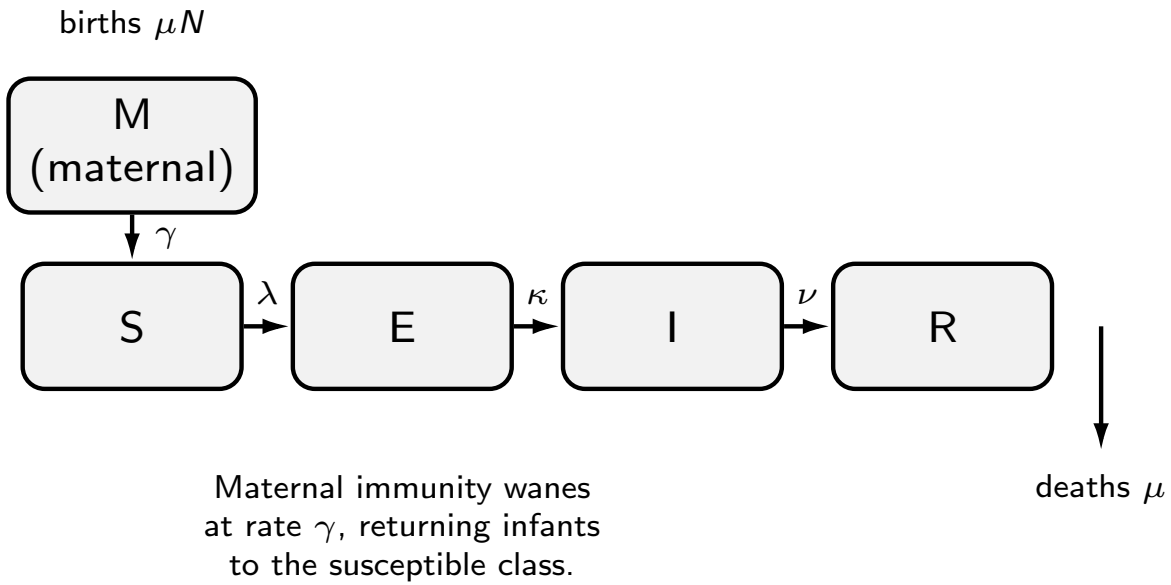
Interpretation:

- β — transmission rate
- κ — progression from latent to infectious
- ν — recovery rate
- μ — birth/death rate
- R_0 depends on both latency and survival during infectious stages

MSEIR Model Structure

Compartments and Interpretation:

- M: infants with **maternal immunity** — protected by antibodies at birth.
- S: **susceptible** individuals — can become infected.
- E: **exposed** — infected but not yet infectious.
- I: **infectious** — capable of transmitting the disease.
- R: **recovered or immune**.
- Maternal immunity wanes at rate γ, moving $M \to S$.
- Births add to M at rate μN; deaths remove from all compartments at rate μ.
- Suitable for diseases such as **measles** or **rubella**, where newborns are initially immune.



MSEIR extends SEIR by accounting for temporary newborn immunity and its loss over time.

Timescales and Qualitative Dynamics in the MSEIR Model

Key Epidemiological Timescales:

- **Maternal immunity:** typically lasts for several **months**.
- **Latent (exposed) period:** a few **days**.
- **Infectious period:** several **days to weeks**.

Implications for Model Behavior:

- Separation of these timescales can lead to:
  - **Oscillatory dynamics** — sustained epidemic cycles (e.g., measles).
  - **Age-structured patterns** — due to slow replenishment of susceptibles.
- The choice of time unit (*days vs years*) influences the numerical behavior of discrete-time models.
- Appropriate scaling ensures model stability and realistic periodicity.

Structured Compartmental Models

Up to now:

- We have explored progressively richer compartmental models:
  - SIR $\rightarrow$ SEIR $\rightarrow$ MSEIR, including births, deaths, and immunity.
  - These models assume **homogeneous mixing** — every individual is equally likely to contact anyone else.

In reality:

- **Contacts are structured** by age, behavior, and setting:
  - Schools, workplaces, and households form distinct sub-networks.
- **Network features** such as degree variation, clustering, and small-world links strongly affect transmission.
- Incorporating structure can change epidemic thresholds, outbreak sizes, and oscillation patterns.

Adding Structure to Epidemic Models

Next step: move from homogeneous populations to **structured mixing**.

- So far, our models assumed **random mixing** — all individuals equally likely to contact each other.
- In reality, transmission depends on:
  - **Age** (children vs adults),
  - **Sex or behavior** (male–female, high–low risk),
  - **Location or community** (schools, workplaces, regions).
- These structures can be captured mathematically by:
  - **Mixing matrices (WAIFW)** — "Who Acquires Infection From Whom,"
  - **Coupled systems** — multiple interacting subpopulations,
  - **Age-structured PDEs** — continuous aging and infection dynamics.

Goal: capture heterogeneity in contact patterns and its effect on transmission.

WAIFW: Who Acquires Infection From Whom

Concept: Represent structured mixing with a **contact matrix** C.

$$C = \begin{pmatrix} \beta_{aa} & \beta_{ab} \\ \beta_{ba} & \beta_{bb} \end{pmatrix}, \qquad \lambda_a = \beta_{aa} I_a + \beta_{ab} I_b, \quad \lambda_b = \beta_{ba} I_a + \beta_{bb} I_b.$$

Interpretation:

- β_{ij} = transmission rate from group j to group i.
- Allows for asymmetric transmission (e.g., children $\rightarrow$ adults differ from adults $\rightarrow$ children).
- Defines the **WAIFW matrix** — "Who Acquires Infection From Whom."
- Foundation for age-, sex-, or behavior-structured models.

Structured contact patterns modify effective R_0 and disease persistence.

Example: SIS Model for Gonorrhea (Heterosexual Contacts)

Assumptions:

- No lasting immunity $\rightarrow$ SIS framework.
- Two populations: females (f) and males (m).
- Transmission occurs only between groups.

Model equations:

$$\frac{dS_f}{dt} = -\beta_{fm} S_f I_m + \nu_f I_f, \qquad \frac{dI_f}{dt} = \beta_{fm} S_f I_m - \nu_f I_f,$$

$$\frac{dS_m}{dt} = -\beta_{mf} S_m I_f + \nu_m I_m, \qquad \frac{dI_m}{dt} = \beta_{mf} S_m I_f - \nu_m I_m.$$

Endemicity condition:

$$R_{0,f} \times R_{0,m} > 1.$$

Implications:

- Gender asymmetry in behavior can sustain infection even if each R_0 individually < 1.
- Useful for modeling sexually transmitted infections (STIs).

Two-Population SIR with Symmetric Coupling

Model:

$$\frac{dS_1}{dt} = -(\beta_{11} I_1 + \alpha I_2)\, S_1 + \mu(1 - S_1),$$

$$\frac{dI_1}{dt} = (\beta_{11} I_1 + \alpha I_2)\, S_1 - (\nu + \mu) I_1,$$

$$\frac{dR_1}{dt} = \nu I_1 - \mu R_1,$$

$$\frac{dS_2}{dt} = -(\beta_{22} I_2 + \alpha I_1)\, S_2 + \mu(1 - S_2),$$

$$\frac{dI_2}{dt} = (\beta_{22} I_2 + \alpha I_1)\, S_2 - (\nu + \mu) I_2,$$

$$\frac{dR_2}{dt} = \nu I_2 - \mu R_2.$$

Key parameter: α — between-group transmission strength.

Dynamics:

- $\alpha = 0$: groups are independent $\rightarrow$ separate epidemics.
- Moderate α: partial synchronization or phase-shifted cycles.
- Large α: synchronized epidemics with shared persistence.

Coupling between populations alters persistence and phase relationships.

Age-Structured SIR Model (PDE Formulation)

Continuous-age formulation:

$$\begin{aligned}
\partial_a S(a,t) + \partial_t S(a,t) &= -[\lambda(a,t) + \mu(a)]S(a,t), \\
\partial_a I(a,t) + \partial_t I(a,t) &= \lambda(a,t)S(a,t) - [\nu + \alpha + \mu(a)]I(a,t), \\
\partial_a R(a,t) + \partial_t R(a,t) &= \nu I(a,t) - \mu(a)R(a,t), \\
S(0,t) &= B(t) \quad \text{(birth boundary condition)}.
\end{aligned}$$

Interpretation:

- Tracks how individuals age and progress through compartments.
- **Characteristics:** (a,t) lines describe cohorts aging over time.
- Computationally intensive but captures realistic demography and immunity patterns.

PDE-based models extend compartmental ideas to continuous age and time structure.

Cohort Models: CAS vs RAS

Purpose: Implement age structure in discrete age bins rather than continuous age–time PDEs.

CAS – Cohort Age-Structured Model:

- ODE system with age classes linked by **aging flow rates** η_i.
- Individuals move from age group i to $i+1$ at rate η_i.
- Continuous aging approximation.

RAS – Realistic Age-Structured Model:

- 1-year age bins; integrate for 1 year, then **shift all cohorts forward**.
- New births enter the age-0 class at each cycle.
- Closer to real census or serological data structures.

Both approaches implement age progression numerically; RAS better matches real-world demographic data.

Beyond Compartmental Models: Agent-Based Modeling (ABM)

Concept: Simulate populations as collections of individual agents with explicit behaviors and interactions.

Key features:

- Each **agent** represents a person (or unit) with state variables (e.g., age, infection status, mobility).
- Agents interact locally according to predefined rules or contact networks.
- The global epidemic emerges from these micro-level interactions.

Advantages:

- Captures heterogeneity in behavior, movement, and compliance.
- Can include spatial structure, stochasticity, and behavioral feedback.
- Useful for policy experiments (e.g., vaccination strategies, school closures).

ABMs go beyond compartmental models by simulating individual-level interactions and emergent epidemic patterns.

Beyond Compartmental Models: Hybrid and Data-Driven Approaches

Hybrid frameworks:

- Combine differential equations with agent-based or network components.
- Multi-scale integration — from within-host dynamics to population spread.

Data-driven methods:

- Statistical inference (Bayesian, particle filters) for parameter estimation.
- Machine learning to predict spread patterns or learn model structure.
- Real-time forecasting using mobility, genomic, or wastewater data.

Challenges:

- Balancing interpretability, accuracy, and computational cost.
- Integrating mechanistic and data-centric paradigms.

Modern infectious disease modeling blends mechanism, data, and computation.

Modeling in Public Health: Classical and Structured Models

Compartmental and structured models remain essential tools for policy planning:

- **Vaccination programs:** optimize timing and coverage (e.g., measles, hepatitis A, COVID-19 boosters).
- **Endemic control:** guide thresholds for elimination and herd immunity ($p_c = 1 - 1/R_0$).
- **Resource allocation:** forecast hospital load or regional outbreaks.
- **Equity and demography:** age-structured and WAIFW models identify vulnerable populations.

Examples:

- National immunization strategies using SEIR frameworks.
- Modeling of maternal immunity for neonatal protection.

Structured compartmental models translate biology into actionable public health policy.

Modeling in Public Health: Modern and Data-Driven Approaches

Beyond compartmental models: new computational frameworks link behavior, data, and policy.

Agent-based models (ABMs):

- Simulate interventions at the individual level — e.g., vaccination campaigns, school closures.
- Capture behavioral adaptation, stochasticity, and spatial heterogeneity.

Hybrid and data-driven approaches:

- Integrate mechanistic models with real-time data streams (mobility, wastewater, genomic surveillance).
- Machine learning for short-term forecasts and uncertainty quantification.
- Coupling with economic and social models for decision support.

Applications:

- COVID-19 response modeling (e.g., mobility-informed SEIR–ABM hybrids).
- Scenario analysis for emerging pathogens and vaccine rollouts.

Modern public health modeling blends mechanistic insight, data, and computation for real-time decision support.

Looking Ahead: Open Challenges in Infectious Disease Modeling

Emerging directions:

- **Data integration:** combining within-host, population, and genomic information.
- **Behavioral feedback:** modeling adaptive human responses and policy effects.
- **Model transparency:** balancing simplicity, realism, and interpretability.
- **Computation:** hybrid deterministic–stochastic and agent-based approaches.
- **Equity and global health:** tailoring models for diverse data environments.

Next generation models will unite biology, computation, and social context.

Ethical Dimensions of Modeling

Why ethics matters:

- Models guide resource allocation and public communication.
- Simplifications may reinforce bias or overlook marginalized populations.
- Uncertainty must be communicated clearly to sustain trust.
- Transparent assumptions foster accountability and reproducibility.

Responsible modeling is not only accurate—but fair and transparent.

Key Takeaways

- **Mathematical modeling provides a lens** to understand, predict, and control infectious disease dynamics.
- **Compartmental frameworks (SIR, SEIR, MSEIR)** capture essential mechanisms — infection, recovery, immunity — and yield interpretable metrics like R_0 and herd immunity thresholds.
- **Structured models** (vital dynamics, age, mixing matrices) refine realism and guide policy for vaccination, equity, and endemic control.
- **Beyond compartmental models** — agent-based, network, and hybrid data-driven approaches — simulate behavior, spatial spread, and real-time responses.
- **Ethical and societal considerations** are integral: transparency, uncertainty, and inclusivity build trust in model-based decisions.

From equations to impact — models translate understanding into informed, equitable public health action.

Thank You!

Questions or Discussion?

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"From equations to impact — the art of modeling infectious diseases."