

Comparative Analysis of Single-Population Compartment Epidemiological Models

Introduction and Overview of the Models

The present work analyses 21 single-population, deterministic, continuous-time compartment ODE models from the perspective of mathematical epidemiology. The aim is to identify connections between the structural properties of each model and its mathematical behaviour: how the existence of an endemic equilibrium, the form of R_0 and the type of bifurcation are influenced by the selection of compartments, the type of the infection term or whether the population is open or closed.

The analysis proceeds in two steps. First, a symbolic script based on the MATLAB Symbolic Toolbox determines the equilibrium points, R_0 , local stability and the Bendixson–Dulac criterion for each model. The symbolic results are complemented by a numerical verification script that uses simulation to check the existence of equilibrium points, the type of bifurcation, and the presence of periodic orbits. The scripts and the result file are publicly available in the following [shared folder](#). The analysed models are shown in the table below, and the results are presented in a criterion-based structure in the remainder of the document.

Group	Models	Key structural feature	Source
Core	SI, SIS, SIR, SIRS, SEIR, SEIS	immunity structure	[1, 2]
Demographic	SIR/SIS/SEIR dem.	open population	[1]
Nonlinear	SIS/SIR saturating, SIS std. inc.	infection term	[1, 3]
Vaccination	SEIRS, SIR vacc., SIRS vacc.	control	[1]
Extended	SIRD, SEIRD, MSIR, SIS-2, SEIR-2, SIQR	extra compartment	[1, 4]

1 Model Structure

1.1 Number and Type of Compartments

The number and biological meaning of state variables determine the dimension of the phase space.

Possible values and their interpretation:

- **2 compartments (S, I):** No recovery (SI) or recovery without immunity (SIS). Reducible to 1D.
- **3 compartments (S, I, R):** Permanent immunity (SIR, SIRS), latency (SEIS), or a demographic variant.
- **4 compartments:** Latency + immunity (SEIR, SIRS vacc.), mortality (SIRD), maternal immunity (MSIR), or quarantine (SIQR). Reducible to 3D.
- **5 compartments:** SEIRD (latency + mortality), SEIR-2 (two infectious stages).
- **4 compartments, two groups (2×2):** SIS-2: R_0 is the spectral radius of a matrix.

The key question is whether the extra compartment feeds back into S or not.

1.2 Closed vs. Open Population

The sum $\sum_i \dot{x}_i$ measures whether the total population size changes over time, where x_i is the size of each compartment (e.g. S, I, R) and \dot{x}_i is its rate of change. If the sum is zero, every individual who leaves one compartment enters another, so N is constant (closed population), if it is nonzero, births or deaths change the total population size (open population).

Possible values and their interpretation:

- **Closed population** ($\sum \dot{x}_i = 0$): $N = S + I + \dots = \text{const}$. The conservation law allows one equation to be eliminated, reducing the system dimension. Suitable for modelling short-term outbreaks. Includes: all core and extended models, except the demographic group and **MSIR**.
- **Open population (demography: rate μ)**: Births (μN) and deaths (μ) both appear. Suitable for modelling long-term endemic diseases. Demography enables the existence of an endemic equilibrium (EE) where a closed population would have none (e.g. **SIR dem.** vs. **SIR**).

This is one of the most important binary structural differences: the same compartment structure exhibits radically different long-term behaviour in an open vs. closed population.

1.3 Type of Infection Term

The mathematical form of the term describing the $S \rightarrow I$ transition determines the nature of the nonlinearity in the infection dynamics and the dependence of R_0 on N .

Possible values and their interpretation:

- **Mass action: βSI** The number of contacts is proportional to the size of both groups. $R_0 = N\beta/(\gamma + \dots)$ — **depends on N** . Assumes an unrealistically large number of contacts in large populations. Applicable: most core and demographic models.
- **Standard incidence: $\beta SI/N$** Each individual has a fixed number of daily contacts, independent of N . $R_0 = \beta/\gamma$ — **independent of N** . Applicable: **SIS std. inc.**, **SIS-2**.
- **Saturating (Holling II): $\beta SI/(1 + \alpha I)$** The contact rate saturates at large I — behavioural feedback. R_0 at the DFE is identical to the mass-action case, but the saturating term can cause **backward bifurcation**. Applicable: **SIS sat.**, **SIR sat.**

The key distinction between mass action and standard incidence is whether the population size enters the epidemic threshold condition.

2 Equilibrium Points

Two types of equilibrium are relevant: the **disease-free equilibrium** (DFE) where $I^* = 0$, and the **endemic equilibrium** (EE) where $I^* > 0$.

2.1 Disease-Free Equilibrium (DFE)

The DFE is the state in which the disease is absent from the population. It exists in every model and typically takes the form $(S^*, I^*) = (N, 0)$.

Possible values:

- **$(N, 0, \dots)$ in a closed population:** All individuals are susceptible. E.g. **SIR**: $(N, 0, 0)$.
- **$(N, 0, \dots)$ in an open population:** Formally identical, but the Jacobian has a different structure (μ terms appear), so the stability analysis differs.
- **$(N(1-p), 0, \dots)$ in a vaccination model:** The effective susceptible population is reduced by the proportion p vaccinated. E.g. **SIR vacc.**: $S_{\text{DFE}}^* = N(1-p)$.

2.2 Existence of the Endemic Equilibrium (EE)

The EE is the state in which $I^* > 0$: the disease persists in the population.

Possible values and their interpretation:

- **No EE exists (closed, no feedback into S):** The R compartment acts as a one-way sink — the disease disappears in the long run. Models: **SIR**, **SIRD**, **SEIRD**, **SIQR**, **SEIR-2**, **SIR sat.**
- **EE exists if $R_0 > 1$ (feedback from recovery):** If recovered individuals return to S ($I \rightarrow S$ or $R \rightarrow S$), the disease is sustainable. Closed models: **SIS**, **SIRS**, **SEIS**, **SEIRS**, **SIS sat.**,

SIS std. inc., SIRS vacc., SIS-2.

- **EE exists if $R_0 > 1$ (demographic feedback):** In an open population, newborns continuously replenish the susceptible pool. Models: SIR dem., SIS dem., SEIR dem., MSIR.
- **EE may exist even if $R_0 < 1$ (backward bifurcation):** Possible in principle with a saturating infection term. Affected: SIS sat. — must be verified manually.

The structural rule: a feedback arc into $S \Leftrightarrow$ EE is possible. Demography ($\mu N \rightarrow S$) and post-recovery return ($\gamma I \rightarrow S$ or $\xi R \rightarrow S$) both count as such feedback.

Type of feedback	Population	Examples	EE?
None	closed	SIR, SIRD, SEIRD, SIQR	no
From recovery ($I \rightarrow S$)	closed	SIS, SEIS	yes
From immunity loss ($R \rightarrow S$)	closed	SIRS, SEIRS	yes
From vaccination	closed	SIRS vacc.	yes
From demography ($\mu N \rightarrow S$)	open	SIR/SIS/SEIR dem.	yes

3 The Basic Reproduction Number (R_0)

R_0 is the average number of new infections caused by a single infectious individual in a fully susceptible population at the very beginning of an epidemic. $R_0 = 1$ is the bifurcation threshold: the disease spreads if $R_0 > 1$ and dies out if $R_0 < 1$. It is computed via the next-generation matrix method [3]:

$$R_0 = \rho(\mathcal{F}\mathcal{V}^{-1}).$$

3.1 Possible Formula Structures for R_0

Base case — mass action, closed, no E compartment.

$$R_0 = \frac{N\beta}{\gamma + (\text{additional exit terms})} \tag{1}$$

Applies to: SIS, SIR, SIRS, SIS sat., SIR sat. The denominator is extended by every term that accelerates exit from the I compartment:

- **$+\mu$** (demography): SIR dem., SIS dem. $\Rightarrow R_0 = N\beta/(\gamma + \mu)$
- **$+\delta$** (mortality): SIRD $\Rightarrow R_0 = N\beta/(\gamma + \delta)$
- **$+\kappa$** (quarantine): SIQR $\Rightarrow R_0 = N\beta/(\gamma + \kappa)$
- **Vaccination (proportion p):** SIR vacc. $\Rightarrow R_0 = N\beta(1 - p)/(\gamma + \mu)$

Every additional exit rate adds to the denominator — mortality and quarantine appear in a mathematically equivalent way.

E compartment with $\mu = 0$ does not change R_0 (it only delays the epidemic): $R_0^{\text{SEIR}} = N\beta/\gamma$.

E compartment with $\mu > 0$: the probability of the $E \rightarrow I$ transition is $\sigma/(\sigma + \mu) < 1$, giving:

$$R_0^{\text{SEIR dem.}} = \frac{N\beta\sigma}{(\sigma + \mu)(\gamma + \mu)}$$

This is the only case in which the E compartment modifies R_0 .

Standard incidence: $R_0 = \beta/\gamma$ — independent of N .

Two-stage SEIR-2: $R_0 = N\beta_1/\delta + N\beta_2/\gamma$ — two additive terms, each stage contributing separately.

SIS-2: R_0 is the spectral radius of the 2×2 next-generation matrix — no simple closed-form formula.

For SEIR-type models (SEIR, SEIS, SEIRD, SEIRS, SEIR-2, SEIR dem.) the symbolic script returns $R_0 = 0$ — the next-generation decomposition must be performed manually.

4 Local Stability Analysis

Local stability analysis examines whether the system, upon receiving a small perturbation around an equilibrium point, returns to it or moves away. The sign of the eigenvalues of the Jacobian matrix determines the outcome, by the Hartman–Grobman theorem the local behaviour of the nonlinear system coincides with that of its linearisation (provided no eigenvalue has zero real part).

Possible values and their interpretation:

- **All $\text{Re}(\lambda_i) < 0 \Rightarrow$ locally stable:** The DFE is locally stable if $R_0 < 1$, and the EE (if it exists) is locally stable if $R_0 > 1$. This holds for every model under study outside the $R_0 = 1$ threshold.
- **At least one $\text{Re}(\lambda_i) > 0 \Rightarrow$ unstable:** The DFE is unstable if $R_0 > 1$ — the disease spreads.
- **Zero real-part eigenvalue ($\text{Re}(\lambda_i) = 0$):** Appears at the $R_0 = 1$ threshold — a bifurcation point at which the Hartman–Grobman theorem does not apply.

λ_{\max} is the eigenvalue of the DFE Jacobian with the largest real part, when $R_0 < 1$ we have $\lambda_{\max} < 0$, and $|\lambda_{\max}|$ determines the rate of exponential decay of perturbations. It typically takes the form $\lambda_{\max} = \gamma(R_0 - 1)$. The structure of local stability is very similar across all models under study, the difference lies in the number and nature (real vs. complex) of the eigenvalues. Complex eigenvalues give rise to oscillatory convergence towards the equilibrium, while real eigenvalues yield monotone convergence.

5 Global Stability Analysis

Global stability examines whether solutions starting from every initial condition within the invariant region of the phase space converge to the equilibrium. The method consists of finding a Lyapunov function $V \geq 0$ for which $\dot{V} \leq 0$ holds throughout the phase space.

Possible values and their interpretation:

- **Globally stable (Lyapunov function found):** The candidate $V = I$ works in many models when $R_0 < 1$: $\dot{V} = \dot{I} = \beta SI - \gamma I = I(\beta S - \gamma) \leq 0$ whenever $S \leq N$ and $R_0 \leq 1$. Holds for: **SIS, SIR, SIRS, SIR dem.** and most 2D models.
- **Global stability open (not proved):** In higher-dimensional models (4–5D) the Lyapunov candidate does not work straightforwardly.
- **Not globally stable (bistability / backward bif.):** With a saturating infection term a stable EE may persist even when $R_0 < 1$. Affected: **SIS sat.**

Global stability analysis depends strongly on the dimension of the system: for 2D models the Bendixson–Dulac criterion and simple Lyapunov candidates are generally sufficient, whereas for higher-dimensional models (4–5D) the construction of an appropriate Lyapunov function is non-trivial.

6 Type of Bifurcation at $R_0 = 1$

Bifurcation analysis examines how the number, coordinates, and stability of equilibrium points change at the threshold value $R_0 = 1$ as a function of the system parameters. The precise type of bifurcation is determined via the centre manifold theorem, which reduces the system to the invariant manifold associated with the zero eigenvalue appearing at $R_0 = 1$.

Possible values and their interpretation:

- **Transcritical bifurcation:** The base case: the DFE and EE exchange stability at $R_0 = 1$. $R_0 < 1$: DFE stable, EE negative (biologically meaningless), $R_0 > 1$: EE positive and stable,

DFE unstable. No bistability. Characteristic of almost all models under study.

- **Backward (saddle-node) bifurcation:** A stable EE may persist even when $R_0 < 1$ — bistability arises. This means that reducing R_0 below 1 may not be sufficient on its own to eliminate the disease. Affected: **SIS sat.** — must be verified manually.
- **Hopf bifurcation:** A periodic solution may arise. Not documented for any of the 21 models, but cannot be ruled out in 3D+ models.

Backward bifurcation appears only with a saturating infection term — this is one of the main motivations for distinguishing infection term types.

7 Periodic Orbits

The analysis of periodic orbits addresses the question of whether the system admits solutions that trace closed paths in the phase space, i.e. whether the epidemic dynamics exhibit oscillatory, cyclic behaviour in the long run. The method is the Bendixson–Dulac criterion: if there exists $B(S, I) > 0$ such that $\nabla \cdot (B\mathbf{f})$ has constant sign, then no periodic orbit exists. The criterion is applicable only to 2D systems.

Possible values and their interpretation:

- **Periodic orbit excluded (Bendixson–Dulac applicable):** With $B = 1/(SI)$ one obtains $\nabla \cdot (B\mathbf{f}) = -\gamma/S^2 < 0$ — no periodic orbit. Holds for: **SIS, SIS dem., SIS sat., SIS std. inc.**, and generally for every 2D model where the right-hand side is sufficiently smooth.
- **Cannot be excluded in 2D (SI):** In the absence of a recovery term the divergence is not of constant sign, so the Dulac criterion does not apply.
- **Not guaranteed in 3D+ models:** The Poincaré–Bendixson theorem applies only in 2D, in higher-dimensional models the existence of periodic orbits must be checked numerically.

8 Reduced Dimension and Final Size

8.1 Reduced Dimension

The conservation law ($\sum x_i = N$) allows the original d -dimensional system to be reduced to a $(d - 1)$ -dimensional one.

Possible values:

- **1D: SI, SIS** — the equation $I = N - S$ suffices.
- **2D: SIR, SIRS, SIS dem.** etc.
- **3D: SEIR, SEIRS, SIRD** etc.
- **Not reducible (open model):** In demographic models N is not constant, so the simple conservation law does not apply directly.

8.2 Final Size (S_∞/N)

The final size is the proportion of individuals never infected by the end of the epidemic — meaningful only for closed models with permanent immunity after recovery.

Possible values:

- **Closed implicit equation:** For **SIR**, $S_\infty = S_0 e^{-R_0(1-S_\infty/N)}$ — solvable numerically.
- **Trivial:** For **SI**, $S_\infty = 0$.
- **Not defined:** In open models and models with an EE — the long-run state is the endemic equilibrium.

Summary

The analytical criteria of the 21 models under study are not independent of one another, they are linked by structural relationships: a single modelling choice simultaneously affects the existence of equilibrium points, the form of R_0 , the type of bifurcation, and the limits of analytical tractability.

The present analysis is restricted to single-population, deterministic, continuous-time models. Natural directions for extension include vector-borne and metapopulation models, models with imperfect vaccines, stochastic variants, network models, and delay differential equations.

References

- [1] Herbert W. Hethcote. The mathematics of infectious diseases. *SIAM Review*, 42(4):599–653, 2000. doi: 10.1137/S0036144500371907.
- [2] William O. Kermack and Anderson G. McKendrick. A contribution to the mathematical theory of epidemics. *Proceedings of the Royal Society of London. Series A*, 115(772):700–721, 1927. doi: 10.1098/rspa.1927.0118.
- [3] Pauline van den Driessche and James Watmough. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical Biosciences*, 180(1–2):29–48, 2002. doi: 10.1016/S0025-5564(02)00108-6.
- [4] James M. Hyman and Jia Li. An intuitive formulation for the reproductive number for the spread of diseases in heterogeneous populations. *Mathematical Biosciences*, 167(1):65–86, 2000. doi: 10.1016/S0025-5564(00)00025-0.