

Master's Project Final Draft

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1 Introduction

"Don't get the flu shot, it'll give you the flu. When the covid vaccine first rolled out, the government was going to track you with a microchip and starve your family by not letting you go to the grocery store. But I did get the vaccine for my family though - didn't let my wife and kids get it - I took one for the team so they could eat."

— unknown

The purpose of this project is to explore how the epidemic-like spread of health misinformation within social networks interacts with the spread of related diseases. While it is always possible for a vaccine to be harmful, or to have other undesirable effects, generally speaking vaccines in wide circulation achieve their intended beneficial purpose and rumors about them causing autism (for example) have never been substantially backed up. The original paper that started this myth was in fact debunked. This paper focuses on the interactions between propagating diseases and related misinformation by using deterministic compartmental models. For simplicity's sake, we generally will assume the vaccines given are effective. We note the more general concept of coupling simultaneous compartmental models can be similar to our work herein, and some discussion on this is given.

A procedure is discussed that can be adapted to use for merging a broad range of compartmental models on a fixed population. We refer to the results of this process as *coupled models* or *coupled state equations*, and the models and states used to construct the coupled models are called *component models* and *component states*. We introduce the procedure under the assumption that state transitions that occur in more than one component state at a time do so with null probability, and later discuss ways to weaken this assumption.

This work was inspired by that of Govindankutty et al., whose SEDPNR model we explain and make use of below.

In an important sense epidemic models and information flow models are the same. Both involve taking stochastic partitions of large populations and approximating their densities with solutions to deterministic ODEs.

Single-layer epidemiological models and information-layer models share a common mathematical language—compartmental flows, transition rates, and reproduction

numbers—but they differ fundamentally in their interpretation, mechanisms, and sources of heterogeneity.

Single-layer disease models describe the flow of infection via contacts between individuals of a population. Generally these are first order systems of ordinary differential equations with mostly linear or bilinear terms in state densities contributing to time derivatives of other state densities. A major assumption of these models is a Markovian transmission process. Most models also assume the population is sufficiently large and homogeneous. Information models function with similar assumptions. There are factors like emotional responses to the information that are considered in these models that typically are not considered in epidemic ones. Both models represent the interaction between susceptible and exposed groups, but diseases are spread through homogeneous physical contact (which contributes terms like βSI to the infection growth rate $\dot{I}$) (Heffernan et al., §2; Ganguly et al., Eq. 1), whereas information flow occurs in asymmetric networks (Mehaa Bhatta 2023, Sec. 2.1–2.4 (often exhibiting power law behavior (Govindankutty Gopalan 2024)) and is considered more sensitive to human agency, with models having compartments representing "Doubters" or "Uninterested" groups or "Positive" or "Negative" emotional valency (Govindankutty Gopalan 2023, Govindankutty Gopalan 2024). Markovian transitions are still the basis of the use of deterministic ODEs to model state densities, but information flow being sensitive to manipulation by algorithms and the ability of humans to learn and adapt given new information in light of old are reasons to consider non-Markovian transitions [Non-Markovian transitions and reinforcement appear in SEDIS (behavioral selection) and Sci. Rep. 2024 (emotional persistence)]. Both disease and information models can be analyzed with a *basic reproduction number* — the average number of new infected (with the disease or information) produced by a typical infected individual (in a completely susceptible population) over its entire lifetime (Heffernan et al., §1). A value of $R_0 > 1$ indicates an epidemic (Heffernan et al., §1). Birth rates and death rates can be added to either model or they will exhibit a conservation of mass. When considering how the phenomena of disease and information spread might interact there are a number of things to consider:

1. Misinformation may lead to behaviors such as vaccine refusal that will alter the rate of vaccination in certain subgroups. Likewise being infected, there being a large or rapidly growing infected subpopulation, or even the death rate of infected people will likely effect how people engage with misinformation. GOVINDANKUTTY shows that misinformation spreads more rapidly when negative emotions are involved. Govindankutty Gopalan 2024. Misinformation impacts behavior, including vaccine hesitancy (Sci. Rep. 2024; Bhatta 2023).
2. Recovering from a disease usually affords a term of immunity from reinfection, whereas this may not be the case for misinformation. [Biological immunity vs. reversible belief states (SEDIS 2023; Maki–Thompson summary).]
3. Feedback loops are possible in information sharing networks which simply do not make sense for disease spread. [Echo chambers and emotional amplification (Sci. Rep. 2024).]
4. Disease processes operate days–weeks; social media operates minutes–hours (Sci. Rep. 2024; SEDIS 2023 observations on rapid digital spread).

The main result of this project is a coupled model $SIR \times SEDPNR^\pm$ with disease-information interaction terms. We show this interaction using the basic reproduction number of the coupled model. We include a proof the model is stable in the sense of Lyapunov, bounded, and positive. We include numerical experiments to illustrate the interaction between the component models.

2 Mathematical Background Overview

2.1 Compartmental Models: deterministic foundations

Given a population Π with N individuals we can impose a partition $P_1 \cup \dots \cup P_k = \Pi$ of the population into disjoint subsets referred to as *states*. The population size may change, as may the state densities, but the partition will remain well defined for all N ; for example we may consider individuals with various age groups. We then can model the dynamics of the population over time with reference to Π using difference equations. Let $x_{i,N} = \#P_i / \#\Pi$ be the proportion of individuals in state P_i be a rational valued function of $t > 0$ for each $i = 1, 2, \dots, k$. We refer to the $x_{i,N}$ as *state proportions*, and naturally they sum to unity $\sum_i x_{i,N}$. We want to use calculus to study these dynamics, but we need to do some work first. We start by setting up a system of difference equations. Let $\delta t > 0$ represent a discrete time step that satisfies $\lim_{N \rightarrow \infty} \delta t = 0$. Define $\delta x_{i,N}(t) = x_{i,N}(t + \delta t) - x_{i,N}(t)$ and define the *transition rate* from state P_i to P_j

$$\rho_{ij}^N(t_0) = \lim_{t \rightarrow t_0^+} \frac{\Pr(\mathcal{E}_t(b) | \mathcal{E}_{t_0}(a))}{t - t_0},$$

where we let $\mathcal{E}_t^N(\sigma)$ be the event that a typical individual of the population is chosen and is in state σ . We then define our equations.

$$\delta x_{i,N}(t) = - \sum_{j \neq i} \rho_{ij}^N(t) x_i^N(t) + \sum_{j \neq i} \rho_{ji}^N(t) x_j^N(t)$$

This will converge to the solution to the differential equation

$$\dot{x}_i(t) = - \sum_{j \neq i} \rho_{ij}(t) x_i(t) + \sum_{j \neq i} \rho_{ji}(t) x_j(t)$$

where $\rho_{ij}^N(t)$ converges to $\rho_{ij}(t)$.

Theorem 2.1. *Let x_N be the vector of solutions to the difference equation. Then, For all $T > 0$, let $f_N(t; T)$ be the function*

$$f_N(t; T) = \sup_{0 \leq t \leq T} \|x_N(t) - x(t)\|$$

where $x(t)$ is the solution to the continuous version of the problem. Then, for $T > 0$ fixed, we have $f_N(t; T)$ converging in probability to 0.

Let $\mathcal{E}_t(\sigma)$ be the event (or rather a limiting case of such an event as $N \rightarrow \infty$) that a typical individual of the population is chosen in state σ . Let $t_0 > 0$. The transition

rate $\rho_{ab}(t_0)$ between two states a and b in a finite state system is understood as an asymptotic version of

$$\rho_{ab}(t_0) = \lim_{t \rightarrow t_0^+} \frac{\Pr(\mathcal{E}_t(b) | \mathcal{E}_{t_0}(a))}{t - t_0}$$

as the population size $N \rightarrow \infty$.

2.2 The basic reproduction number

The basic reproduction number of a network epidemic model is “the expected number of secondary infections arising from a single infected individual during his or her entire infectious period, in a wholly susceptible population.” (Heffernan, Smith, Wahl 2005, §1) [?]. The number is denoted R_0 .

Definition 2.2. Consider a population that is host to an epidemic. Let $F(\tau)$ be the probability that a newly infected individual remains infectious for at least time $t = \tau$, and let $b(\tau)$ be the average number of new infections an infectious individual will produce per unit time after being infected for time $t = \tau$. Then the *basic reproduction number* of the epidemic is

$$\int_0^\infty F(t)b(t)dt$$

R_0 provides a measure of spreadability of a contagion. It is a threshold quantity that distinguishes between invasion and extinction:

- If $R_0 < 1$, each infected individual produces less than one new infection on average, and the disease cannot invade.
- If $R_0 > 1$ the infection can invade and persist. (Heffernan et al. 2005, p. 2–3)

Historically, versions of this quantity appear already in the work of Kermack–McKendrick (1927) and in demographic studies of Lotka and Dublin (1925). See Dietz (1993) for a historical overview.

Most prevalent in modern literature is the Markovian *Next-Generation Matrix* formulation of R_0 . For compartmental ODE systems of the form¹

$$\dot{x} = \mathcal{F}(x) - \mathcal{V}(x)$$

where $\mathcal{F}$ represents new infection terms and $\mathcal{V}$ contains all other terms, the *next generation matrix* is $K = FV^{-1}$ where $F = D\mathcal{F}(x^*)$ and $V = D\mathcal{V}(x^*)$ are given by linearization at the *disease-free equilibrium* x^* , i.e. the natural equilibrium with all infected state densities vanishing. The basic reproduction number is then $R_0 = \rho(K)$, the spectral radius of the next-generation matrix. This is the dominant modern framework, introduced rigorously by Diekmann, Heesterbeek Metz (1990) and clarified further in Mathematical Tools for Understanding Infectious Disease Dynamics (Diekmann, Heesterbeek Roberts 2010)[?]. Heffernan–Smith–Wahl (2005, §2.2) present this formulation as the natural extension of the survival-function approach when multiple infectious states exist.

¹typically what is done in practice is to look at a truncated system with only the infected states considered as variables

Renewal (Non-Markovian) Formulation

The renewal formulation provides a general construction of the basic reproduction number in settings where the infectious period or the infectivity profile need not follow exponential (Markovian) waiting-time distributions. In such models, the time since infection—the *infection age*—governs both infectiousness and the possibility of remaining infectious. Let $a \geq 0$ denote the infection age.

Two functions determine the transmission process:

1. The *survival probability* $F(a)$, defined as the probability that an infected individual remains infectious at least until infection age a . This function is not assumed to be exponential, and no Markov property is required.
2. The *infectivity function* $b(a)$, defined as the average rate at which an individual of infection age a generates new infections in an otherwise susceptible population.

The basic reproduction number is given by the total expected production of secondary infections over the entire infectious lifetime:

$$R_0 = \int_0^\infty b(a) F(a) da. \quad (1)$$

This expression represents the lifetime offspring number of a single infection and gives us a solid definition of R_0 for general deterministic epidemic models [?, ?]. It applies equally well to structured systems, vector-borne transmission cycles, and models with arbitrary infectious-period distributions.

The same functions $b(a)$ and $F(a)$ generate the renewal equation for the incidence. If $i(t)$ denotes the incidence at time t , then near the disease-free equilibrium one has the Volterra-type integral equation

$$i(t) = \int_0^\infty i(t-a) b(a) F(a) da, \quad (2)$$

which requires no explicit compartmental structure. Assuming early exponential growth of the form $i(t) = e^{rt}$ yields the characteristic equation

$$1 = \int_0^\infty b(a) F(a) e^{-ra} da, \quad (3)$$

first formalized in the general theory of next-generation operators [?]. The parameter r denotes the Malthusian growth rate of the infection. The classical threshold condition

$$R_0 > 1 \quad \Longleftrightarrow \quad r > 0$$

holds without modification, establishing that the renewal formulation preserves the standard invasion criterion even in the absence of Markovian assumptions.

The non-Markovian renewal approach is therefore essential in modelling epidemic processes with realistic infectiousness profiles, including infections with strong age-of-infection effects, multiple latent stages, peaked infectious periods, behavioural changes, or general waiting-time distributions. It provides the most fundamental expression of the basic reproduction number in such settings.

2.3 Stability Theory for Compartmental Systems

The stability properties of equilibria in compartmental epidemic models are classically analyzed using Lyapunov methods. This subsection summarizes the main concepts and the reasons these tools apply so effectively to Metzler population–balance systems such as SIR, SEIR, SEDIR, and their coupled extensions.

Lyapunov stability vs. global asymptotic stability. Let $\dot{x} = f(x)$ be a smooth dynamical system with equilibrium x^* . Following [?, ?, ?], the equilibrium x^* is called *Lyapunov stable* if, for every $\varepsilon > 0$, there exists $\delta > 0$ such that $\|x(0) - x^*\| < \delta$ implies $\|x(t) - x^*\| < \varepsilon$ for all $t \geq 0$. It is *asymptotically stable* if it is Lyapunov stable and $\lim_{t \rightarrow \infty} x(t) = x^*$ for all sufficiently small perturbations. If these properties hold for all initial conditions, the equilibrium is *globally asymptotically stable* (GAS).

In population models, GAS of the disease-free equilibrium is tightly linked to threshold parameters such as the basic reproduction number R_0 [?, ?].

Lyapunov functionals for epidemic systems. Compartmental epidemic models are positive systems whose Jacobians are Metzler matrices on the positively invariant region $\mathbb{R}_+^n$. For such systems, it is often possible to construct simple Lyapunov functionals, usually of the form

$$V(x) = \sum_i c_i x_i \quad \text{or} \quad V(x) = x^\top P x,$$

with $c_i > 0$, $P \succeq 0$, chosen so that $\dot{V}(x) \leq 0$ holds on the feasible region. This classic method appears in Murray–Sastry [?] and in the control-theoretic expositions of [?, ?]. For epidemic models possessing a DFE, V is typically designed to decrease monotonically whenever $R_0 < 1$, yielding global stability of the DFE.

Comparison theorems and Metzler systems. Because epidemic models are order-preserving (cooperative) systems with Metzler Jacobians, comparison principles apply directly: if $x(t)$ satisfies $\dot{x} \leq g(x)$ and $y(t)$ satisfies $\dot{y} = g(y)$ with $x(0) \leq y(0)$, then $x(t) \leq y(t)$ for all t . Such monotonicity results allow Lyapunov functions to be combined with upper/lower bounding systems. This is central in the analysis of next-generation matrices and the relationship between R_0 and stability [?, ?].

Why global Lyapunov functions exist for many epidemic models. Compartmental models used in epidemiology are

- (i) *positively invariant* on a compact simplex (the population fractions sum to 1);
- (ii) *cooperative* (off-diagonal partials are nonnegative);
- (iii) governed by a *Metzler* linearization at the DFE; and
- (iv) often *dissipative*.

These structural properties ensure that simple convex Lyapunov functions (e.g. weighted sums of compartments) frequently suffice to establish GAS of the DFE when $R_0 < 1$, and to rule out spurious equilibria in the feasible region. This phenomenon is discussed in [?, ?, ?, ?] and is one reason Lyapunov methods remain a standard tool for epidemic stability analysis.

3 The SEDPNR Information Model

To model the spread of misinformation on a social network, we adopt a six-compartment framework originally introduced by Govindankutty, Velappan, and Kuttichira [?]. The model tracks the evolution of individuals through the information states

$$S \longrightarrow E \longrightarrow D \longrightarrow \{P, N\} \longrightarrow R,$$

where each transition represents a distinct psychological or behavioral step in the process of rumor or misinformation diffusion.

Our model has the same dynamics as they introduce, but we note that our interpretation of the states is slightly different since the application we make of it is our own. In the final version of our model we actually split the restrained compartment into two separate ones as we will see below.

Compartment meanings. Following [?], the six compartments are:

- **S (Susceptible).** Individuals who have not yet encountered the misinformation.
- **E (Exposed).** Individuals who have seen the claim but have not formed a judgment about its validity.
- **D (Doubter).** Individuals who are undecided and seek verification; they may move toward either belief or denial.
- **P (Positive Spreader).** Individuals who spread the information with a positive emotional valence (e.g. happy or excited about it)
- **N (Negative Spreader).** Individuals who spread the information with a negative emotional valence (e.g. afraid or angry)
- **R (Restrained).** Individuals who become uninterested, fatigued, or otherwise disengaged, and cease participating in the information spread.

The positive and negative states we will replace with misinformation propagators and information negators. This will then lead us to split the restrained state into two according to what spreading/belief state they were in before disengaging. This is done because even though an individual may cease spreading the misinformation, their opinion on it will still influence their behavior vis a vis vaccinations.

Transitions. In the original SEDPNR model [?], the allowed transitions are:

$$\begin{aligned} S &\xrightarrow{\alpha} E, & E &\xrightarrow{\gamma} D, & E &\xrightarrow{\mu_1} S, & D &\xrightarrow{\mu_2} S, \\ D &\xrightarrow{\beta_1} P, & D &\xrightarrow{\beta_2} N, & P &\xrightarrow{\lambda} R, & N &\xrightarrow{\lambda} R. \end{aligned}$$

These transitions distinguish SEDPNR from classical rumor models (e.g. Daley–Kendall or Maki–Thompson) by introducing both a “doubter” stage and a split between positive and negative engagement. In their paper, the authors show the significance of the emotional valence of a post toward its spreadability.

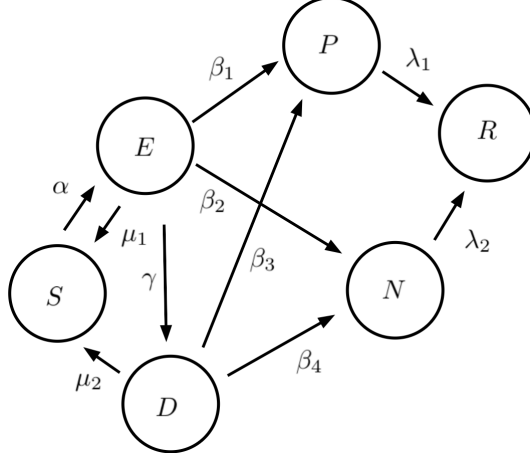


Figure 1: Illustration of the SEDPNR model.

Interpretation of the rates.

- α : rate at which individuals first encounter the rumor.
- γ : rate at which exposed individuals actively seek clarification and become doubters.
- μ_1, μ_2 : “forgetting” or “loss of interest” rates, returning individuals to the susceptible class.
- β_1 : rate at which doubters become positive spreaders.
- β_2 : rate at which doubters become negative spreaders.
- λ_1, λ_2 : rate at which active participants (either P or N) lose interest and become restrained.

Deterministic ODE formulation.

Let

$$S(t), E(t), D(t), P(t), N(t), R(t)$$

denote the respective population fractions. The SEDPNR dynamics of [?] are then:

$$\dot{S} = -\alpha S + \mu_1 E + \mu_2 D, \quad (4)$$

$$\dot{E} = \alpha S - (\gamma + \mu_1) E, \quad (5)$$

$$\dot{D} = \gamma E - (\mu_2 + \beta_1 + \beta_2) D, \quad (6)$$

$$\dot{P} = \beta_1 D - \lambda_1 P, \quad (7)$$

$$\dot{N} = \beta_2 D - \lambda_2 N, \quad (8)$$

$$\dot{R} = \lambda_1 P + \lambda_2 N. \quad (9)$$

4 A basic viral model

We introduce here an SEIR system to illustrate viral applications before we discuss coupling models.

$$\begin{aligned}\dot{S} &= -\beta SI, \\ \dot{E} &= \beta SI - \sigma E, \\ \dot{I} &= \sigma E - \gamma I, \\ \dot{R} &= \gamma I,\end{aligned}$$

with DFE $(S, E, I, R) = (1, 0, 0, 0)$ in normalized units, i.e. where we have $S + E + I + R = 1$ and our variables represent proportions not raw numbers.

Interpretation of the rates.

- β : this is the rate at which contacts with infected individuals produce new infections.
- σ : this is the rate at which exposed individuals become infected/contagious.
- γ : this is the rate at which individuals become no longer able to spread the disease after infection.

This is an easy example for computing the basic reproduction number of an epidemic. The infected subsystem is (E, I) . Near the DFE:

$$F = \begin{pmatrix} \beta & 0 \\ 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \sigma & 0 \\ -\sigma & \gamma \end{pmatrix}.$$

Thus,

$$R_0 = \rho(FV^{-1}) = \frac{\beta}{\gamma},$$

and we can see intuitively how the number R_0 is related to viral spread in this case quite easily.

5 The coupled model

5.1 Introduction to coupling

The overall goal is to analyze compartmental models that arise from taking a version of cartesian products of the directed weighted graphs of given compartmental models. For a pair of graphs, their *cartesian product* is the graph whose vertex set is the cartesian product of the original graphs' and where a pair of paired vertices has an edge between them in the new graph if exactly one of the component vertex pairs is an edge in the corresponding original graph. By interpreting the symmetric incidence relations defining adjacency of vertices as elements of a boolean algebra and then tossing in the weights (positive functions of time) with the appropriate direction (plus or minus sign), we obtain a starting place for describing how many phenomena that can be modeled with compartmental models on the same population interact.

There are significantly fewer possibly non-vanishing transition rates in any potential coupled model if we make the assumptions that make this construction natural than if we were to approach coupling the models from examining each pair of paired states individually. With certain assumptions on the phenomena modeled, this modified cartesian approach is valid.

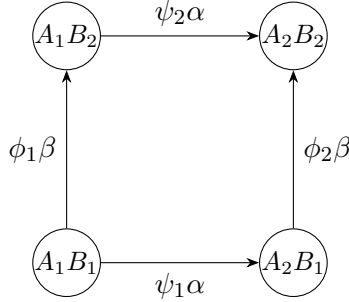
Example 1. We will begin with an example of how two models' equations can be coupled. Consider two component models on a fixed population with two states each.

$$\alpha : A_1 \rightarrow A_2 \quad \beta : B_1 \rightarrow B_2 \quad (10)$$

Let x_1, x_2 denote the A state proportions and y_1, y_2 denote the B state proportions. The equations for these models are below.

$$\begin{aligned} \dot{x}_1 &= -\alpha x_1 & \dot{y}_1 &= -\beta y_1 \\ \dot{x}_2 &= \alpha x_1 & \dot{y}_2 &= \beta y_2 \end{aligned} \quad (11)$$

We also will assume that for a fixed component, the coupled transition rates are scaled by a function of that fixed index. This is illustrated below where ϕ and ψ are functions of t that depend on the phenomena modeled.



The equations of the models A and B have the matrix forms $M_A x = \dot{x}$ and $M_B y = \dot{y}$ and so does our new coupled model. Notice that we must select an ordered basis for our new state space to get a unique matrix and we use the ordering $\Lambda : 11 < 12 < 21 < 22$.

$$\dot{z} = M_{A \otimes B}^\Lambda z = \begin{pmatrix} -\phi_1 \beta - \psi_1 \alpha & 0 & 0 & 0 \\ \phi_1 \beta & -\psi_2 \alpha & 0 & 0 \\ \psi_1 \alpha & 0 & -\phi_2 \beta & 0 \\ 0 & \psi_2 \alpha & \phi_2 \beta & 0 \end{pmatrix} \begin{pmatrix} z_{11} \\ z_{12} \\ z_{21} \\ z_{22} \end{pmatrix}$$

We would get a different but equivalent matrix if we choose the ordering $\Lambda' : 11 < 21 < 12 < 22$ for example, namely the one below.

$$M_{A \otimes B}^{\Lambda'} = \begin{pmatrix} -\phi_1 \beta - \psi \alpha & 0 & 0 & 0 \\ \psi \alpha & -\phi_2 \beta & 0 & 0 \\ \phi_1 \beta & 0 & -\psi_2 \alpha & 0 \\ 0 & \phi_2 \beta & \psi_2 \alpha & 0 \end{pmatrix}$$

While their order is different, the diagonals will of course be the same since those represent the outflow terms.

Example 2. Consider a two state system $S : A_1 \rightarrow A_2$ and a three state system $T : B_1 \rightarrow B_2, B_2 \rightarrow B_3$.



The equations for S and T are $S : Ax = \dot{x}$ and $T : By = \dot{y}$ for some matrices A and B . We can think of the coupled model as having variables z_{ij} that are the entries of a matrix Z . The equations of the coupled model can then be written easily using our assumptions on how we couple them, and the matrices A and B . We have the equations for the coupled model in the following.

$$S \otimes T : \dot{Z} = AZ\Psi + \Phi ZB^T$$

The matrices Φ and Ψ are the modifiers from above

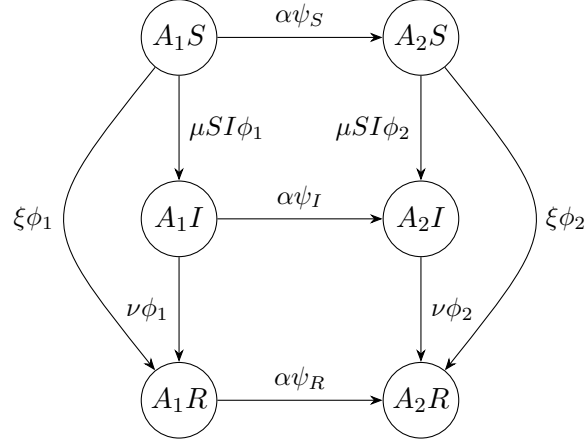
Example 3. Now we will show a less abstract example. One of the models is an SI model of the spread, in a social network, of misinformation about a viral vaccine being ineffective or harmful (we assume the vaccine to in fact be effective). The second is an SIR model for the virus for which the vaccine is being administered. We are assuming all members of the population that are able to become infected by the virus also participate in the social network.



The component models have the following equations. We are somewhat confusingly using the notation SIR for the variables of the model and the labels of the states, but this is commonly done for this model in the literature. When it is helpful we will employ the symbols y_i (for $i = 1, 2, 3$ taken in the natural order) instead of the SIR ones. The variable x_1 represents proportion of social network users not exposed to the information yet, and x_2 represents same for the exposed users.

$$\begin{aligned} \dot{x}_1 &= -\alpha x_1 & \dot{S} &= -\mu SI - \xi S \\ \dot{x}_2 &= \alpha x_1 & \dot{I} &= \mu SI - \nu I \\ & & \dot{R} &= \xi S + \nu I \end{aligned}$$

We will begin with a fairly general setup for how we couple the equations and explain assumptions we make along the way. We will assume just as we did in the previous example that diagonal transitions in the coupled model are negligible, and that having a fixed state in one component has a uniform effect on the overall transition rate with respect to changes in the other component. Let $\phi = (\phi_1, \phi_2)$ and $\psi = (\psi_S, \psi_I, \psi_R)$ be non-negative functions of t . The coupled equations will be built from the network model below.



5.2 Component Models and State Space

We consider two directed graphs:

- a *viral layer* with vertices

$$V(G_v) = \{S_v, I_v, R_v, V_v\},$$

representing susceptible, infected, recovered, and vaccinated individuals in a classical SIRV framework; and

- an *information layer* with vertices

$$V(G_{\text{info}}) = \{S, E, D, P, N, R^+, R^-\},$$

corresponding to an SEDPNR[±] compartmental model for health misinformation.

The information layer tracks individuals who are

- S : unaware or indifferent to the rumor,
- E : exposed but not yet committed,
- D : doubters who have seen the content but remain unconvinced,
- P : positive sharers (promoting accurate information),
- N : negative sharers (spreading misinformation),
- R^+ : retired but positively inclined,
- R^- : retired but negatively inclined.

The viral layer tracks:

- S : Uninfected individuals
- I : infected/contagious individuals
- R : individuals that recovered from the virus after catching it and are now immune
- V : individuals who get the vaccine and are now immune

Interpretation of the information rates.

- α : rate at which individuals first encounter the rumor.
- γ : rate at which exposed individuals actively seek clarification and become doubters.
- μ_1, μ_2 : “forgetting” or “loss of interest” rates, returning individuals to the susceptible class.
- β_1 : rate at which doubters become believers/propagators.
- β_2 : rate at which doubters become negators.
- λ_1 : rate at which believers lose interest in actively sharing the information
- λ_2 : rate at which deniers lose interest in actively sharing/combating the information

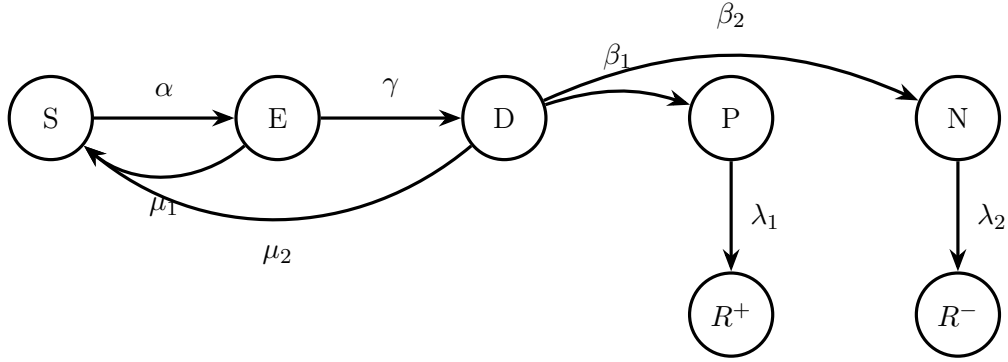


Figure 2: Information-layer SEDPNR $^\pm$ component graph with improved label spacing.

We order the viral compartments as (S_v, I_v, R_v, V_v) and include natural demography and disease-induced mortality in the linear generator. Let

- $\xi > 0$ denote the per-capita vaccination rate $S_v \rightarrow V_v$,
- $\zeta > 0$ the per-capita recovery rate $I_v \rightarrow R_v$,
- $\Omega > 0$ the natural mortality rate (acting on all classes),
- $\omega \geq 0$ the excess infectious mortality multiplier, so that infectives die at rate $(1 + \omega)\Omega$.

Births at rate B are collected in the inhomogeneous vector b and do not appear in the generator itself, and the nonlinear infection term is handled separately in the incidence function and therefore is not included in Q_v .

In this ordering we take

$$Q_v = \begin{pmatrix} -(\xi + \Omega) & 0 & 0 & 0 \\ 0 & -(\zeta + (1 + \omega)\Omega) & 0 & 0 \\ 0 & \zeta & -\Omega & 0 \\ \xi & 0 & 0 & -\Omega \end{pmatrix},$$

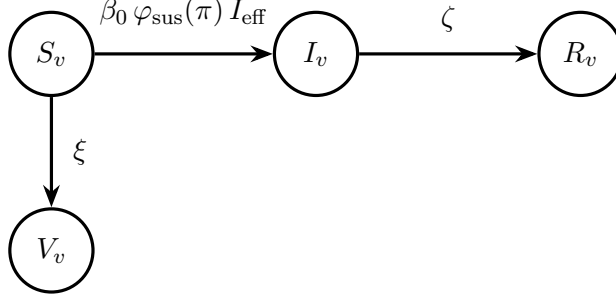


Figure 3: Viral-layer SIRV component graph with clear spacing of transition rates.

so that, for column vectors $X_v(t) = (S_v(t), I_v(t), R_v(t), V_v(t))^\top$, the linear part of the uncoupled viral dynamics satisfies

$$X'_v(t) = Q_v X_v(t) + b, \quad b = (B, 0, 0, 0)^\top$$

when demography is included.

The viral and information layers are combined by taking the Cartesian product digraph

$$G = G_v \square G_{\text{info}}.$$

A vertex of G is a pair (x_i, y_j) with $x_i \in V(G_v)$ and $y_j \in V(G_{\text{info}})$; this pair is interpreted as “current viral status x_i and current information status y_j .”

At the level of population densities, we collect the states into a 4×7 matrix

$$Z(t) = [z_{ij}(t)]_{i=1,\dots,4}^{j=1,\dots,7},$$

ordered so that the rows are (S_v, I_v, R_v, V_v) and the columns are $(S, E, D, P, N, R^+, R^-)$. Thus, for instance, $z_{24}(t)$ is the density of individuals who are virally infected and are positive sharers of information at time t .

We write the row vectors

$$X^S(t), X^I(t), X^R(t), X^V(t) \in \mathbb{R}^7$$

for the first, second, third, and fourth rows of $Z(t)$ respectively. These encode the information-layer compositions within each viral class.

5.3 Uncoupled Generator on the Product

Before introducing the nonlinear feedback terms, it is convenient to describe the linear generator associated to the Cartesian product $G_v \square G_{\text{info}}$.

Let Q_v be the 4×4 generator of the SIRV model (including natural death and demography, if present), and let Q_{info} be the 7×7 generator of the SEDPNR $^\pm$ subsystem, ordered by $(S, E, D, P, N, R^+, R^-)$. In matrix form we take

$$Q_{\text{info}} = \begin{pmatrix} -\alpha & \mu_1 & \mu_2 & 0 & 0 & 0 & 0 \\ \alpha & -(\mu_1 + \gamma + \beta_1 + \beta_2) & 0 & 0 & 0 & 0 & 0 \\ 0 & \gamma & -(\mu_2 + \beta_3 + \beta_4) & 0 & 0 & 0 & 0 \\ 0 & \beta_1 & \beta_3 & -\lambda_1 & 0 & 0 & 0 \\ 0 & \beta_2 & \beta_4 & 0 & -\lambda_2 & 0 & 0 \\ 0 & 0 & 0 & \lambda_1 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \lambda_2 & 0 & 0 \end{pmatrix}^\top,$$

so that $X'(t) = Q_{\text{info}}X(t)$ for column vectors X .

Using the standard Kronecker-sum representation, the uncoupled linear dynamics for the vectorized $Z(t)$ are

$$\dot{z}(t) = (Q_v \otimes I_7 + I_4 \otimes Q_{\text{info}}) z(t) + b,$$

for a suitable birth vector b if demography is included. When we group rows instead of vectorizing, this induces linear operators L^S, L^I, L^R, L^V acting on the block rows X^S, X^I, X^R, X^V . This comes in handy in coding the numerical algorithm later on and makes for much more readable equations. The full set of equations is included in the appendix.

5.4 Information Composition and Effective Infection Signal

We now introduce the *information composition* of the susceptible viral class. Let

$$X^S(t) = (X_1^S(t), \dots, X_7^S(t))^\top$$

be the row of $Z(t)$ corresponding to S_v . Its total mass is

$$v_S(t) = \sum_{j=1}^7 X_j^S(t),$$

and we define the normalized composition

$$\pi_j(t) = \frac{X_j^S(t)}{v_S(t)}, \quad j = 1, \dots, 7,$$

whenever $v_S(t) > 0$. In particular, we denote

$$\pi_S, \pi_E, \pi_D, \pi_P, \pi_N, \pi_{R^+}, \pi_{R^-}$$

for the components of the vector $\pi(t)$.

To capture the idea that certain information states are more effective at transmitting infection, we introduce a nonnegative weight vector

$$\sigma = (\sigma_1, \dots, \sigma_7)^\top,$$

and define the *effective infection signal*

$$I_{\text{eff}}(t) := \sigma^\top X^I(t) = \sum_{k=1}^7 \sigma_k X_k^I(t).$$

An alternative choice, which we briefly discuss in the conclusion, is to take $I_{\text{eff}}(t) = \sum_j \theta_j X_j^I(t)$ with the same coefficients θ_j that appear in the susceptibility modulation below.

5.5 Information-Dependent Modifiers

We use the information composition to modulate viral infection and vaccination rates.

5.5.1 Susceptibility modifier

We first define a scalar susceptibility response

$$\phi_{\text{sus}}(\pi_P, \pi_N) \in [0, \infty),$$

which depends on the total weight in positive and negative sharers and incorporates a simple polarization penalty. Given parameters $\kappa > 0$, $r \in [-1, 1]$, and $p \geq 0$, we set

$$u = \pi_P + \pi_N, \quad m = \pi_P - \pi_N,$$

$$a_0 = 1 - \frac{\kappa}{4}(1 + r)^2,$$

and

$$\phi_{\text{sus}}(\pi_P, \pi_N) = a_0 + \kappa r u + \kappa u(1 - u) - p a_0 m^2.$$

The term $u(1 - u)$ rewards moderate but not overwhelming focus on sharers, while the negative term $-p a_0 m^2$ penalizes heavily polarized mixtures where one of π_P, π_N dominates the other.

We assign a susceptibility modifier $c_j(\pi)$ to each information state via

$$c_j(\pi) = 1 + \theta_j \phi_{\text{sus}}(\pi_P, \pi_N),$$

where $\theta_j \geq 0$ controls the strength of the response of class j . Collecting these into a diagonal matrix gives

$$C(\pi) = \text{diag}(c_1(\pi), \dots, c_7(\pi)).$$

5.5.2 Vaccination modifier

We also allow vaccination effectiveness to depend on the information composition through a response function

$$\phi_{\text{vax}}(\pi_E, \pi_D, \pi_P, \pi_{R+}).$$

Given parameters β_2, β_4, κ , we set

$$\phi_{\text{vax}}(\pi_E, \pi_D, \pi_P, \pi_{R+}) = \beta_2 \pi_E + \beta_4 \pi_D + \pi_P + \kappa \pi_{R+}.$$

Here π_P and π_{R+} increase trust in vaccination, while π_E and π_D (weighted by β_2, β_4) reflect the drag from uncertain or skeptical individuals.

With a baseline vaccination rate $v_0 > 0$ and the same heterogeneity vector $\theta = (\theta_1, \dots, \theta_7)$, we define

$$v_j(\pi) = v_0(1 - \theta_j \phi_{\text{vax}}(\pi_E, \pi_D, \pi_P, \pi_{R+})),$$

and set

$$V(\pi) = \text{diag}(v_1(\pi), \dots, v_7(\pi)).$$

The $v_j(\pi)$ encode how quickly individuals in information class j move from viral susceptibility to vaccination in response to the global information composition.

5.6 Modified Information Exposure

Within the information layer, we single out the basic transition $S \rightarrow E$ (raw exposure to the rumor). Let E be the 7×7 matrix with a single nonzero entry corresponding to this transition, so that EX inserts the flow from S to E .

We then define modified exposure operators for the infected and recovered viral blocks:

$$A^{(I)} := (1 + \eta)\alpha E, \quad A^{(R)} := (1 - \rho)\alpha E,$$

with parameters $\eta > 0$ and $\rho > 0$. The idea is that infected individuals are slightly more likely to be exposed to content about the disease, while recovered individuals are slightly less likely.

5.7 Block Form of the Coupled System

Collecting the information above, the coupled system can be written at the level of the 7-vectors X^S, X^I, X^R, X^V . The uncoupled linear part is

$$\dot{X}^S = L^S X^S, \quad \dot{X}^I = L^I X^I, \quad \dot{X}^R = L^R X^R, \quad \dot{X}^V = L^V X^V,$$

where each L^j encodes the SEDPNR $^\pm$ generator acting on information states, together with possible demographic terms.

The information-modulated perturbations are then added as

$$\dot{X}^S = L^S X^S - C(\pi) X^S I_{\text{eff}} - V(\pi) X^S, \quad (12)$$

$$\dot{X}^I = L^I X^I + C(\pi) X^S I_{\text{eff}} + A^{(I)} X^I, \quad (13)$$

$$\dot{X}^R = L^R X^R + A^{(R)} X^R, \quad (14)$$

$$\dot{X}^V = L^V X^V + V(\pi) X^S. \quad (15)$$

Here

$$\pi = \frac{X^S}{\sum_{j=1}^7 X_j^S}, \quad I_{\text{eff}} = \sigma^\top X^I,$$

and the matrices $C(\pi)$, $V(\pi)$, $A^{(I)}$, $A^{(R)}$ depend on the parameters of the chosen scenario.

Equations (12)–(15) are equivalent to the 4×7 matrix formulation

$$\dot{Z} = \hat{Q}(Z) + \mathcal{N}(Z),$$

where $\hat{Q}$ is the linear part induced by Q_v and Q_{info} , and $\mathcal{N}(Z)$ collects the nonlinear infection and vaccination terms built from $C(\pi)$, $V(\pi)$, and I_{eff} .

5.8 Lyapunov Functionals Adapted to the Coupled System

In this section we outline Lyapunov functionals for the coupled SIRV $\times$ SEDPNR $^\pm$ model introduced above. We work on the positively invariant region

$$\mathcal{D} := \left\{ (X^S, X^I, X^R, X^V) \in (\mathbb{R}_{\geq 0}^7)^4 : X^S + X^I + X^R + X^V = N \right\},$$

where $N > 0$ is the total population size. Recall that the information composition is

$$\pi(t) = \frac{X^S(t) + X^I(t) + X^R(t) + X^V(t)}{N},$$

and that the coupled dynamics can be written in block form as

$$\begin{aligned}\dot{X}^S &= L^S X^S - C(\pi) X^S I_{\text{eff}} - V(\pi) X^S, \\ \dot{X}^I &= L^I X^I + C(\pi) X^S I_{\text{eff}} + A^{(I)} X^I, \\ \dot{X}^R &= L^R X^R + A^{(R)} X^R, \\ \dot{X}^V &= L^V X^V + V(\pi) X^S.\end{aligned}$$

5.9 Disease-free equilibrium and next-generation matrix

The *disease-free equilibrium* (DFE) is the state

$$X^I = 0, \quad X^R = 0, \quad X^V = X^{V,0}, \quad X^S = X^{S,0},$$

with $X^{S,0} + X^{V,0} = N$ and information composition $\pi^0 = (1, 0, \dots, 0)$ concentrated in the S -information class. Linearizing the infected block X^I around the DFE gives

$$\dot{X}^I = (F - V)X^I,$$

where F collects new infection terms and V collects transitions out of infectious classes (recovery, vaccination, and death). The basic reproduction number R_0 is defined as

$$R_0 = \rho(FV^{-1}),$$

where $\rho(\cdot)$ denotes the spectral radius.

5.10 Lyapunov functional for the viral subsystem

We first construct a Lyapunov functional on the viral subsystem, treating the information dynamics as frozen at the DFE $\pi = \pi^0$ and $I_{\text{eff}} = 0$. Let $X^I \in \mathbb{R}_{\geq 0}^7$ denote the infected densities ordered by information state. Following the standard next-generation approach, assume that V is a nonsingular M-matrix and set

$$H := V - F.$$

When $R_0 < 1$, H is an M-matrix with all eigenvalues having negative real part. There exists a positive diagonal matrix $P_v = \text{diag}(p_1, \dots, p_7)$ such that the symmetric part

$$S_v := P_v H + H^\top P_v$$

is negative definite. We define

$$V_{\text{virus}}(X^I) := (X^I)^\top P_v X^I. \tag{16}$$

Then $V_{\text{virus}} \geq 0$ on $\mathbb{R}_{\geq 0}^7$, $V_{\text{virus}} = 0$ iff $X^I = 0$, and along trajectories of the linearized viral subsystem one has

$$\dot{V}_{\text{virus}} = (X^I)^\top S_v X^I \leq 0,$$

with strict negativity away from $X^I = 0$.

5.11 Lyapunov functional for the information subsystem

We now isolate the information subsystem by considering the pure SEDPNR[±] dynamics with health state fixed in the susceptible layer. Let

$$Y := X^S \in \mathbb{R}_{\geq 0}^7$$

and write the information-only dynamics in the form

$$\dot{Y} = L^{\text{info}} Y,$$

where L^{info} is the 7×7 generator obtained from the full model by setting $\nu = \zeta = \xi = 0$ and restricting to the susceptible viral class.

Under the assumptions:

- (I1) the non-diagonal entries of L^{info} are nonnegative (birth–death type structure);
- (I2) the row sums of L^{info} are nonpositive, and at least one row sum is strictly negative;

L^{info} is the generator of an irreducible Markov chain with absorbing state in the R^+ and R^- classes. As before, there exists a positive diagonal matrix $P_{\text{info}} = \text{diag}(w_1, \dots, w_7)$ such that the symmetric part

$$S_{\text{info}} := P_{\text{info}} L^{\text{info}} + (L^{\text{info}})^{\top} P_{\text{info}}$$

is negative semi-definite. We define

$$V_{\text{info}}(Y) := Y^{\top} P_{\text{info}} Y. \quad (17)$$

Then $V_{\text{info}} \geq 0$ and $\dot{V}_{\text{info}} \leq 0$, with $\dot{V}_{\text{info}} = 0$ only on the absorbing subspace spanned by R^+ and R^- .

5.12 Combined Lyapunov functional and no-diagonal-jump structure

For the full coupled system we exploit the block structure and the *no diagonal jumps* assumption: there are no transitions that simultaneously change both the viral and information coordinates in a single event. In particular, the Jacobian of the full system at the DFE is block upper triangular in the decomposition

$$(X^I, X^S, X^R, X^V),$$

with infection appearing only in off-diagonal terms that are nonnegative.

Let $a, b > 0$ be constants to be chosen and define

$$V(X^S, X^I, X^R, X^V) := a V_{\text{virus}}(X^I) + b V_{\text{info}}(X^S + X^R + X^V). \quad (18)$$

Because the susceptibility modifier ϕ_{sus} is bounded,

$$0 \leq \phi_{\text{sus}}(u) \leq C_{\text{sus}},$$

the matrices $C(\pi)$ and $V(\pi)$ remain uniformly bounded and their contribution to $\dot{V}$ can be absorbed by choosing a and b appropriately.

Theorem 5.1 (DFE stability for the coupled system). *Assume that:*

- (H1) $R_0 = \rho(FV^{-1}) < 1$ for the viral subsystem;
- (H2) the susceptibility modifier ϕ_{sus} is bounded and Lipschitz continuous;
- (H3) there are no diagonal jumps between viral and information states, so that the full Jacobian at the DFE is block upper-triangular with Metzler blocks;
- (H4) the demographic parameters satisfy $\Omega > 0$, $B > 0$ so that $\mathcal{D}$ is positively invariant.

Then there exist positive constants $a, b > 0$ such that the functional V in (18) is a Lyapunov function for the coupled system at the DFE. In particular, the DFE is globally asymptotically stable in $\mathcal{D}$.

Proof sketch. By construction, $V \geq 0$ on $\mathcal{D}$ and $V = 0$ if and only if $X^I = 0$ and $X^S + X^R + X^V$ lies in the absorbing information subspace. Using the block structure of the Jacobian and the bounds on $C(\pi)$ and $V(\pi)$, one shows that $\dot{V} \leq 0$ along solutions. The strict inequalities inherited from V_{virus} and V_{info} then imply that the largest invariant set contained in $\{\dot{V} = 0\}$ is the DFE. LaSalle's invariance principle yields the claim. $\square$

5.13 Stability of Endemic Equilibria

When $R_0 > 1$ the system may admit one or more endemic equilibria with $X^I \neq 0$. In this regime Lyapunov methods can still be useful, but must be adapted to the nonlinear structure of the infection and information feedback.

5.14 Uniqueness and Jacobian sign structure

Let $x^* = (X^{S,*}, X^{I,*}, X^{R,*}, X^{V,*})$ be an endemic equilibrium. We say x^* is *biologically feasible* if all components are strictly positive and the corresponding information composition π^* lies in the interior of the simplex.

Sufficient conditions for uniqueness of such an equilibrium can be formulated in terms of:

- monotonicity of the effective infection signal $I_{\text{eff}} = \theta^T X^I$;
- sign conditions on the Jacobian $J(x^*)$, such as strict diagonal dominance in the infection–information sub-blocks or the existence of a Metzler–M-matrix decomposition.

Under these hypotheses x^* is the unique endemic equilibrium in $\mathcal{D} \setminus \{\text{DFE}\}$, and one can attempt to construct a Lyapunov functional of Volterra type,

$$V_{\text{end}}(x) := \sum_k c_k \left(x_k - x_k^* - x_k^* \log \frac{x_k}{x_k^*} \right),$$

with coefficients $c_k > 0$ chosen to match the sign structure of $J(x^*)$. The detailed construction and verification of $\dot{V}_{\text{end}} \leq 0$ are deferred to future work.

6 Numerical scenarios using the coupled model

For numerical experiments we implement the coupled system in Python using a classical fourth-order Runge–Kutta (RK4) scheme. Writing $y(t) \in \mathbb{R}^{28}$ for the vectorization of $Z(t)$, and $f(t, y)$ for the right-hand side determined by (12)–(15), one RK4 step of size $h > 0$ from time t_n is

$$\begin{aligned} k_1 &= f(t_n, y_n), \\ k_2 &= f(t_n + \frac{h}{2}, y_n + \frac{h}{2}k_1), \\ k_3 &= f(t_n + \frac{h}{2}, y_n + \frac{h}{2}k_2), \\ k_4 &= f(t_n + h, y_n + hk_3), \\ y_{n+1} &= y_n + \frac{h}{6}(k_1 + 2k_2 + 2k_3 + k_4). \end{aligned}$$

We use a uniform time step and integrate over a fixed time horizon $[0, T]$.

Throughout all the numerical scenarios we *fix the viral parameters* to

$$\nu = 0.5, \quad \zeta = 0.25,$$

so that the infection and recovery rates in the SIRV layer are identical across experiments. In this way, differences in epidemic behavior arise solely from the information-layer transition rates, the heterogeneity weights θ , and the effective infection weights σ .

As a common initial condition we take a population that is already experiencing a modest outbreak:

$$X^S(0) = (0.9, 0, 0, 0, 0, 0, 0), \quad X^I(0) = (0.1, 0, 0, 0, 0, 0, 0),$$

with $X^R(0) = X^V(0) = 0$. Thus 90% of the population is virus-susceptible and 10% is infected, all with information state S at time $t = 0$.

An effective reproduction number

Because the infection term in (13) is nonlinear in the infected profile X^I through I_{eff} , the usual next-generation matrix construction must be adapted. For the purpose of comparing scenarios, we adopt the following linearized effective reproduction number R_0 .

Near the disease-free configuration where all individuals are in (S_v, S) , the information composition in the susceptible class is

$$\pi = (\pi_S, \pi_E, \pi_D, \pi_P, \pi_N, \pi_{R^+}, \pi_{R^-}) = (1, 0, 0, 0, 0, 0, 0).$$

For this composition we have

$$\phi_{\text{sus}}(0, 0) = a_0 = 1 - \frac{1}{4} \kappa (1 + r)^2,$$

so the susceptibility factor in the S column is

$$c_S = 1 + \theta_1 a_0,$$

where θ_1 is the first entry of the heterogeneity vector θ . Approximating I_{eff} by the contribution of the infected mass in S alone, we obtain an effective infection rate

$$\beta_{\text{eff}} \approx \nu c_S \sigma_1,$$

where σ_1 is the first entry of the incidence weight vector σ . We then define

$$R_0 := \frac{\beta_{\text{eff}}}{\zeta} \approx \frac{\nu}{\zeta} c_S \sigma_1 = \frac{\nu}{\zeta} (1 + \theta_1 a_0) \sigma_1. \quad (19)$$

This quantity is the average number of secondary infections generated by an individual in the (I_v, S) state, under the linearization around the (S_v, S) disease-free configuration. Because we hold ν and ζ fixed across scenarios, variations in R_0 are driven entirely by information-layer parameters.

In addition to the total viral time series, we also visualize (i) the information composition $\pi^S(t)$ within the susceptible class S_v and (ii) the scalar signals $I_{\text{eff}}(t)$, $\phi_{\text{sus}}(t)$, and $\phi_{\text{vax}}(t)$ defined from $\pi(t)$ and $X^I(t)$. These plots clarify how information flow modulates infection and vaccination in each scenario.

6.1 Scenario A: Moderately informative environment

In Scenario A, the SEDPNR $^\pm$ dynamics emphasize relatively rapid movement from exposure to either doubting or sharing, but with modest rates of long-term retirement. Representative choices are

$$\begin{aligned} \alpha &= 0.4, & \gamma &= 0.2, & \mu_1 &= 0.2, & \mu_2 &= 0.1, \\ \beta_1 &= 0.3, & \beta_2 &= 0.25, & \beta_3 &= 0.1, & \beta_4 &= 0.05, \\ \lambda_1 &= 0.1, & \lambda_2 &= 0.1. \end{aligned}$$

The effective infection weights σ are chosen so that exposed and doubting classes carry slightly more influence than fully retired classes; for example,

$$\sigma = (0.1, 0.15, 0.15, 0.2, 0.1, 0.15, 0.15).$$

Behavioral parameters such as v_0 , κ , r , p , η , and ρ are chosen at moderate magnitudes so that information feedback modifies, but does not dominate, the SIRV dynamics; the first heterogeneity weight is

$$\theta_1 = 0.1.$$

For this scenario the effective reproduction number (19) evaluates to

$$R_0^{(A)} \approx 0.22,$$

so the epidemic remains subcritical: the infected population gradually decays from its initial 10% level and no large secondary wave appears. The viral time-series plot shows a monotone or gently peaked decline in I_v , accompanied by a steady rise in the vaccinated class V_v .

The information-composition plot for S_v shows that the susceptible population quickly develops a nontrivial mix of exposed and doubting states, but a substantial fraction remains in S and the system slowly drifts into the retired compartments. The behavioral-modifier plot illustrates that $I_{\text{eff}}(t)$ and $\phi_{\text{sus}}(t)$ remain relatively modest throughout, while $\phi_{\text{vax}}(t)$ produces a sustained but moderate vaccination pressure.

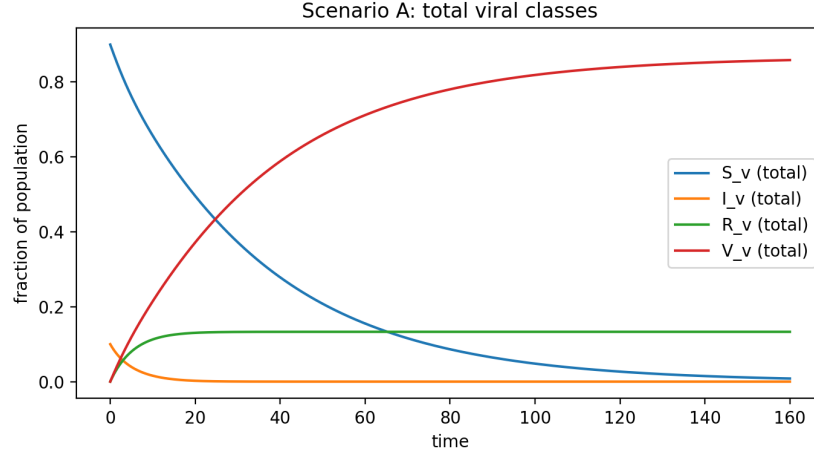


Figure 4: Total viral classes S_v , I_v , R_v , V_v over time in Scenario A.

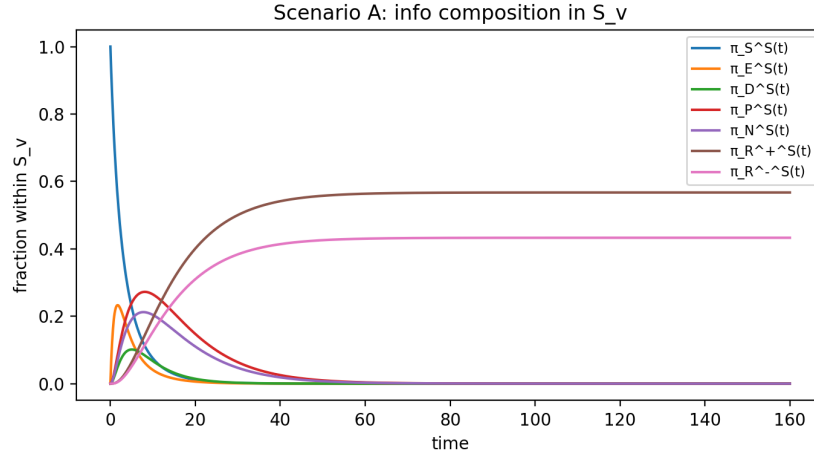


Figure 5: Information composition $\pi^S(t)$ within the susceptible class S_v in Scenario A.

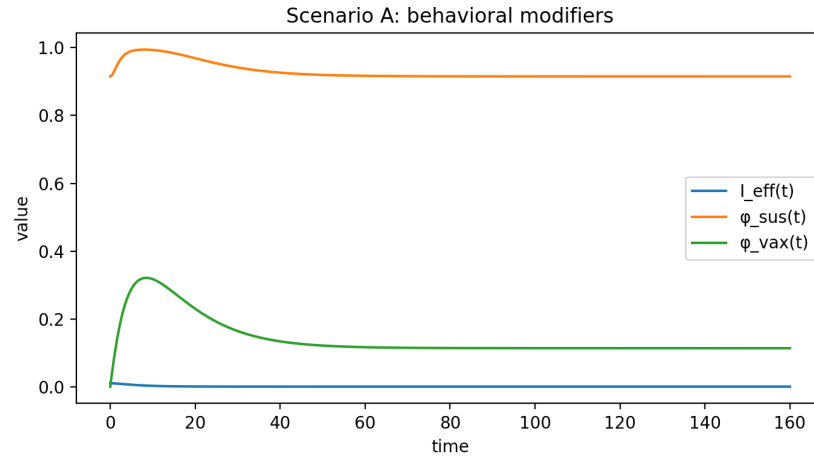


Figure 6: Behavioral signals $I_{\text{eff}}(t)$, $\phi_{\text{sus}}(t)$, and $\phi_{\text{vax}}(t)$ in Scenario A.

6.2 Scenario B: Strong polarization and selective susceptibility

Scenario B increases the strength of transitions into positive and negative sharers,

$$\beta_1 = \beta_3 = 0.4, \quad \beta_2 = \beta_4 = 0.25,$$

and assigns more heterogeneous susceptibility weights

$$\theta = (0.2, 0.3, 0.5, 0.9, 0.9, 0.4, 0.4),$$

with effective infection weights

$$\sigma = (0.1, 0.15, 0.15, 0.2, 0.1, 0.1, 0.2).$$

In this setting, the susceptibility modifier $C(\pi)$ amplifies infection when the susceptible viral class is concentrated in strongly opinionated information states (either positive or negative), and the vaccination modifier $V(\pi)$ reacts more sharply to shifts in π_E , π_D , π_P , and π_{R+} .

The stronger heterogeneity in θ yields a larger first entry $\theta_1 = 0.2$, while σ_1 and (ν, ζ) remain as in Scenario A. The effective reproduction number is therefore slightly larger:

$$R_0^{(B)} \approx 0.24.$$

The epidemic is still subcritical, but the infected population declines more slowly than in Scenario A. The viral time series shows a longer-lived infected tail, with a somewhat delayed transfer of mass into the recovered and vaccinated classes.

The information-composition plot reveals a pronounced transient concentration of the susceptible population in the sharing states P and N , reflecting the stronger flows into those compartments. Correspondingly, the behavioral-modifier plot shows larger excursions in $I_{\text{eff}}(t)$ and $\phi_{\text{sus}}(t)$, indicating that transient polarization can temporarily increase the effective force of infection even when $R_0 < 1$.

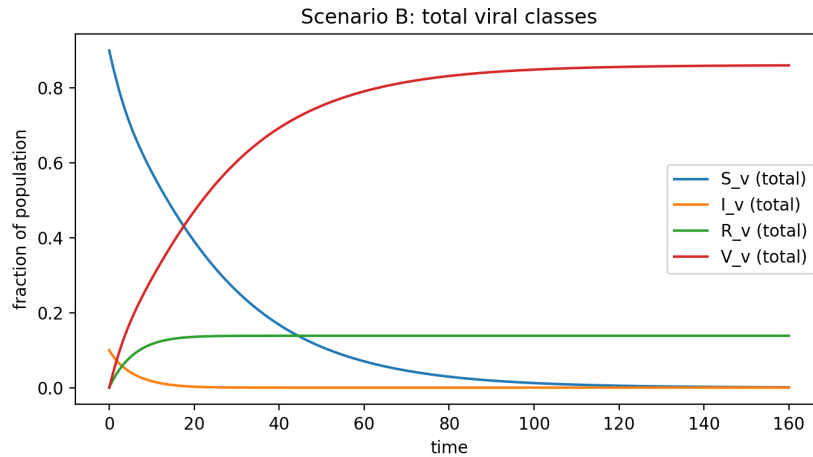


Figure 7: Total viral classes S_v , I_v , R_v , V_v over time in Scenario B.

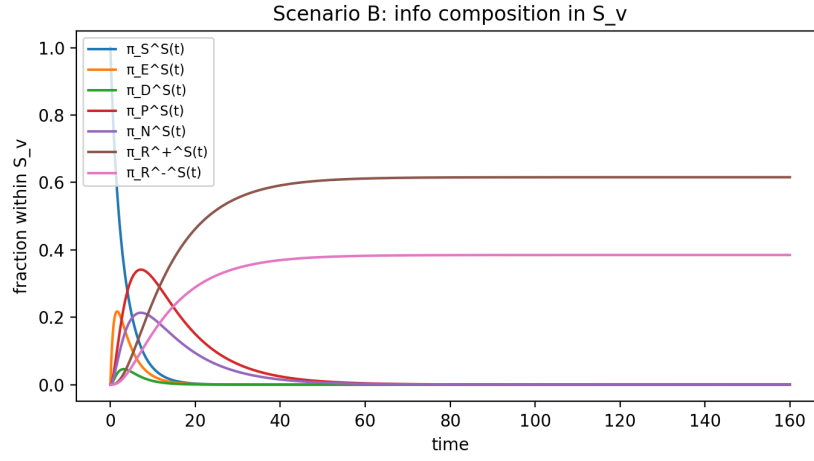


Figure 8: Information composition $\pi^S(t)$ within the susceptible class S_v in Scenario B.

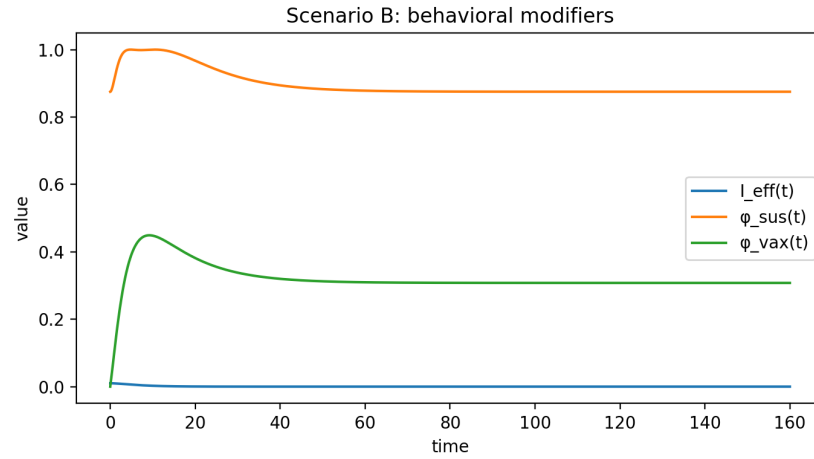


Figure 9: Behavioral signals $I_{\text{eff}}(t)$, $\phi_{\text{sus}}(t)$, and $\phi_{\text{vax}}(t)$ in Scenario B.

6.3 Scenario C: Pro-vaccine bias and strong polarization penalty

In Scenario C we introduce a stronger pro-vaccine bias in the information response and a more pronounced penalty for polarized mixtures of P and N . One illustrative choice is

$$\begin{aligned}\alpha &= 0.4, \quad \gamma = 0.2, \quad \mu_1 = \mu_2 = 0.1, \\ \beta_1 &= 0.45, \quad \beta_2 = 0.3, \quad \beta_3 = 0.35, \quad \beta_4 = 0.2, \\ \lambda_1 &= 0.05, \quad \lambda_2 = 0.1,\end{aligned}$$

with heterogeneity and infection weights

$$\theta = (0.1, 0.2, 0.3, 1.2, 0.8, 0.3, 0.3),$$

$$\sigma = (0.05, 0.05, 0.1, 0.5, 0.1, 0.1, 0.1).$$

We also take a smaller baseline vaccination rate v_0 and stronger nonlinearity parameters r, p, κ in ϕ_{sus} and ϕ_{vax} , reflecting the idea that positive sharers and the positively retired class have disproportionate influence on vaccination decisions, while highly polarized mixtures of P and N are penalized.

In this regime the first entries θ_1 and σ_1 are both relatively small, and the resulting effective reproduction number is

$$R_0^{(C)} \approx 0.11,$$

substantially below one. The viral time series shows a comparatively rapid decline of the infected population and a more aggressive rise in the vaccinated class, driven by the strong pro-vaccine bias in ϕ_{vax} .

The information-composition plot for S_v exhibits a sharp transient rise in P and R^+ , followed by a drift into retired states. The N class remains strongly suppressed, both by the polarization penalty encoded in ϕ_{sus} and by feedback from the infected layer. The behavioral-modifier plot shows a relatively large $\phi_{\text{vax}}(t)$ peak compared to $\phi_{\text{sus}}(t)$, consistent with a regime in which information campaigns primarily accelerate vaccination rather than amplifying infection.

7 Qualitative behavior and outlook

Across all three scenarios the model exhibits plausible qualitative behavior consistent with the effective reproduction numbers $R_0^{(A)}, R_0^{(B)}, R_0^{(C)} < 1$:

- Information feedback through $C(\pi)$ and I_{eff} can slow or accelerate the decay of the infected population, depending on the sign and magnitude of θ_j in the high-weight information states and on the transient distribution of $\pi(t)$ in S_v .
- The vaccination modifier $V(\pi)$ captures how sustained positive messaging can accelerate the movement into V_v , thereby reducing the duration and overall burden of the epidemic even when the basic SIRV parameters (ν, ζ) are fixed.
- Polarization penalties in ϕ_{sus} can prevent the system from locking into strongly negative sharing states and instead push mass into retired compartments, while still allowing transient peaks in $I_{\text{eff}}(t)$ when sharing classes become temporarily dominant.

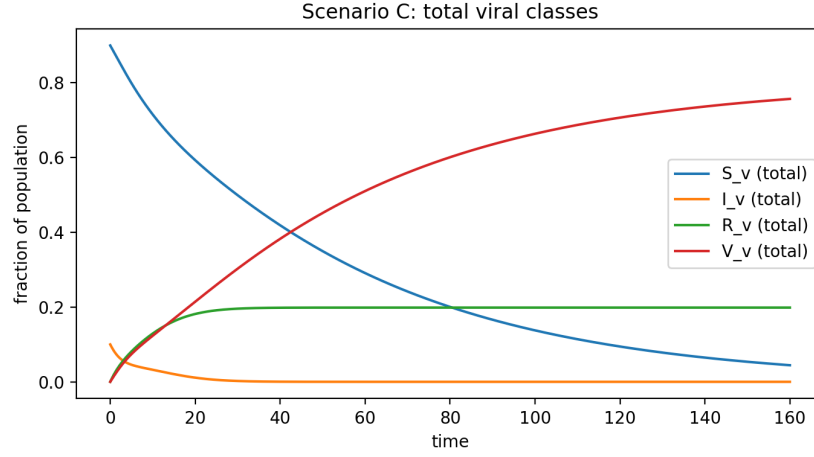


Figure 10: Total viral classes S_v , I_v , R_v , V_v over time in Scenario C.

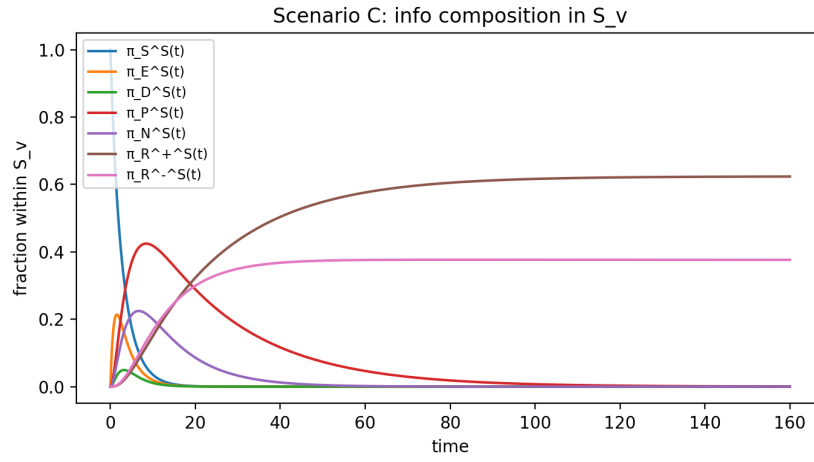


Figure 11: Information composition $\pi^S(t)$ within the susceptible class S_v in Scenario C.

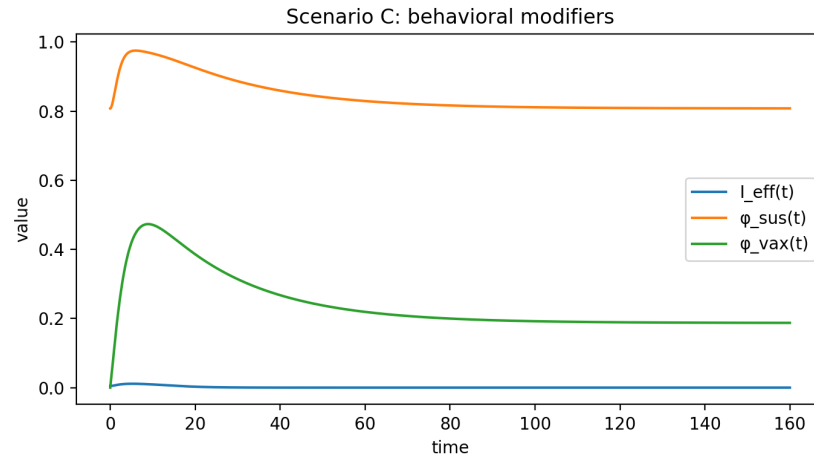


Figure 12: Behavioral signals $I_{\text{eff}}(t)$, $\phi_{\text{sus}}(t)$, and $\phi_{\text{vax}}(t)$ in Scenario C.

The RK4 implementation developed for these experiments can be reused for more elaborate scenarios (for example, adding time-dependent contact rates, non-Markovian dwell-time distributions, or alternative definitions of I_{eff}), and the block structure (12)–(15) makes it straightforward to compare purely linear coupling choices to the nonlinear response functions considered here. The effective reproduction number (19) provides a convenient scalar summary for such comparisons, while the information-composition and behavioral-modifier plots give a more detailed picture of how information and disease co-evolve.

Appendix

This appendix contains the formal justifications for the continuous-time compartmental ODE limit and for the Cartesian-product coupling construction used throughout the thesis. These arguments are standard in the theory of density-dependent Markov population processes, following the classical work of Kurtz [?, ?], and are included here for completeness.

Law of Large Numbers Limit for Discrete Compartment Models

We justify the passage from discrete, finite-population models to deterministic ODE dynamics. Let a population of size N be partitioned at each time t into states $P_1, \dots, P_k$, and let

$$x_i^{(N)}(t) = \frac{1}{N} \# \{\text{individuals in } P_i \text{ at time } t\}.$$

Assume that time evolves in increments $\delta t = \delta t_N > 0$ with $\delta t_N \rightarrow 0$ and $N\delta t_N \rightarrow \infty$ as $N \rightarrow \infty$. During each time step $[t, t + \delta t)$ an individual in state i transitions to $j \neq i$ with probability

$$\Pr(i \rightarrow j \text{ in } \delta t) = \rho_{ij}(t) \delta t + o(\delta t),$$

where the transition rates $\rho_{ij}(t)$ are uniformly Lipschitz and bounded.

Let $\Delta x_i^{(N)}(t) = x_i^{(N)}(t + \delta t) - x_i^{(N)}(t)$. Then

$$\mathbb{E}[\Delta x_i^{(N)}(t) \mid x^{(N)}(t)] = \delta t \left[- \sum_{j \neq i} \rho_{ij}(t) x_i^{(N)}(t) + \sum_{j \neq i} \rho_{ji}(t) x_j^{(N)}(t) \right] + o(\delta t),$$

while the conditional variance satisfies

$$\text{Var}(\Delta x_i^{(N)}(t)) = O\left(\frac{\delta t}{N}\right).$$

Define $x(t)$ to be the solution of the limiting system

$$\dot{x}_i(t) = - \sum_{j \neq i} \rho_{ij}(t) x_i(t) + \sum_{j \neq i} \rho_{ji}(t) x_j(t), \quad i = 1, \dots, k. \quad (20)$$

Theorem .1 (Kurtz-type Law of Large Numbers). *Let $x^{(N)}(t)$ be the density process defined above and $x(t)$ the unique solution to (20). Then for every finite $T > 0$,*

$$\sup_{0 \leq t \leq T} \|x^{(N)}(t) - x(t)\| \xrightarrow[N \rightarrow \infty]{\Pr} 0.$$

Proof sketch. The expected increment of $x^{(N)}$ matches the drift of the ODE system (20). The fluctuations have variance of order $O(\delta t/N)$, so by Chebyshev's inequality they are $o(\delta t)$ uniformly in time. Summing over $T/\delta t$ steps and applying a Grönwall inequality yields convergence in probability. See [?, ?] for the full proof. $\square$

Thus deterministic compartmental ODEs faithfully describe the limit of large, well-mixed populations evolving under small time increments.

Vanishing Probability of Simultaneous Transitions

A central modeling assumption in Section ?? is that *simultaneous transitions* in different model components occur with probability of order dt^2 , and therefore do not contribute to the generator of the limiting continuous-time process.

Suppose an individual can undergo a viral transition A at rate λ and an information transition B at rate α . Over a short time interval $[t, t + dt)$,

$$\Pr(A) = \lambda dt + o(dt), \quad \Pr(B) = \alpha dt + o(dt).$$

Assuming independence at leading order,

$$\Pr(A \cap B) = (\lambda dt)(\alpha dt) + o(dt^2) = O(dt^2).$$

As $dt \rightarrow 0$, these events vanish faster than dt and therefore do not contribute to the infinitesimal generator of the limiting process. The continuous-time model thus retains only transitions in which *exactly one component* changes at a time, matching the edges of the Cartesian product digraph.

Justification of the Cartesian-Product Generator

Let G_v be the viral-layer digraph with generator Q_v , and let G_{info} be the information-layer digraph with generator Q_{info} . For the product state space $V(G_v) \times V(G_{\text{info}})$, define the transition

$$(x, y) \rightarrow (x', y)$$

whenever $x \rightarrow x'$ is a viral transition, and analogously

$$(x, y) \rightarrow (x, y')$$

whenever $y \rightarrow y'$ is an information transition.

By the argument of the previous section, transitions affecting both coordinates simultaneously have probability $O(dt^2)$ and do not appear in the limit. Therefore the generator Q of the joint process must satisfy

$$Q = Q_v \otimes I + I \otimes Q_{\text{info}},$$

the Kronecker sum of the component generators. In the matrix-block notation used in the main text, this is the origin of the block operators L_S, L_I, L_R, L_V and of the linear part of the 28-dimensional system.

The nonlinear infection and vaccination terms introduced in Section ?? act as perturbations of this Kronecker-sum operator but preserve the one-coordinate-at-a-time transition structure required by the continuous-time limit.

Renewal Formulation of the Basic Reproduction Number

For completeness we record the renewal formulation of the basic reproduction number R_0 for non-Markovian epidemic processes. Let $F(a)$ be the probability that an infected individual remains infectious at least until infection age a , and let $b(a)$ be the expected rate of generating new infections at infection age a . Then

$$R_0 = \int_0^\infty b(a)F(a) da.$$

Linearizing the renewal equation for incidence,

$$i(t) = \int_0^\infty i(t-a)b(a)F(a) da,$$

with ansatz $i(t) = e^{rt}$ gives the characteristic equation

$$1 = \int_0^\infty b(a)F(a)e^{-ra} da.$$

The sign of r determines the stability of the disease-free equilibrium, and $r > 0$ is equivalent to $R_0 > 1$. This general framework underlies both the classical and matrix-based definitions of R_0 used in the main text.

Unperturbed coupled equations full

$$SS \quad \dot{z}_{11} = B + \mu_1 z_{12} + \mu_2 z_{13} - (\alpha + \nu + \xi + \Omega) z_{11}, \quad (21)$$

$$IS \quad \dot{z}_{21} = \mu_1 z_{22} + \mu_2 z_{23} + \nu z_{11} - (\alpha + \zeta + \omega\Omega) z_{21}, \quad (22)$$

$$RS \quad \dot{z}_{31} = \mu_1 z_{32} + \mu_2 z_{33} + \zeta z_{21} - (\alpha + \Omega) z_{31}, \quad (23)$$

$$VS \quad \dot{z}_{41} = \mu_1 z_{42} + \mu_2 z_{43} + \xi z_{11} - (\alpha + \Omega) z_{41}, \quad (24)$$

$$SE \quad \dot{z}_{12} = \alpha z_{11} - (\nu + \xi + \mu_1 + \beta_1 + \beta_2 + \gamma + \Omega) z_{12}, \quad (25)$$

$$IE \quad \dot{z}_{22} = \alpha z_{21} + \nu z_{12} - (\zeta + \mu_1 + \beta_1 + \beta_2 + \gamma + \omega\Omega) z_{22}, \quad (26)$$

$$RE \quad \dot{z}_{32} = \alpha z_{31} + \zeta z_{22} - (\mu_1 + \beta_1 + \beta_2 + \gamma + \Omega) z_{32}, \quad (27)$$

$$VE \quad \dot{z}_{42} = \alpha z_{41} + \xi z_{12} - (\mu_1 + \beta_1 + \beta_2 + \gamma + \Omega) z_{42}, \quad (28)$$

$$SD \quad \dot{z}_{13} = \gamma z_{12} - (\nu + \xi + \mu_2 + \beta_3 + \beta_4 + \Omega) z_{13}, \quad (29)$$

$$ID \quad \dot{z}_{23} = \gamma z_{22} + \nu z_{13} - (\zeta + \mu_2 + \beta_3 + \beta_4 + \omega\Omega) z_{23}, \quad (30)$$

$$RD \quad \dot{z}_{33} = \gamma z_{32} + \zeta z_{23} - (\mu_2 + \beta_3 + \beta_4 + \Omega) z_{33}, \quad (31)$$

$$VD \quad \dot{z}_{43} = \gamma z_{42} + \xi z_{13} - (\mu_2 + \beta_3 + \beta_4 + \Omega) z_{43}, \quad (32)$$

$$SP \quad \dot{z}_{14} = \beta_1 z_{12} + \beta_3 z_{13} - (\nu + \xi + \lambda_1 + \Omega) z_{14}, \quad (33)$$

$$IP \quad \dot{z}_{24} = \beta_1 z_{22} + \beta_3 z_{23} + \nu z_{14} - (\zeta + \lambda_1 + \omega\Omega) z_{24}, \quad (34)$$

$$RP \quad \dot{z}_{34} = \beta_1 z_{32} + \beta_3 z_{33} + \zeta z_{24} - (\lambda_1 + \Omega) z_{34}, \quad (35)$$

$$VP \quad \dot{z}_{44} = \beta_1 z_{42} + \beta_3 z_{43} + \xi z_{14} - (\lambda_1 + \Omega) z_{44}, \quad (36)$$

$$SN \quad \dot{z}_{15} = \beta_2 z_{12} + \beta_4 z_{13} - (\nu + \xi + \lambda_2 + \Omega) z_{15}, \quad (37)$$

$$IN \quad \dot{z}_{25} = \beta_2 z_{22} + \beta_4 z_{23} + \nu z_{15} - (\zeta + \lambda_2 + \omega\Omega) z_{25}, \quad (38)$$

$$RN \quad \dot{z}_{35} = \beta_2 z_{32} + \beta_4 z_{33} + \zeta z_{25} - (\lambda_2 + \Omega) z_{35}, \quad (39)$$

$$VN \quad \dot{z}_{45} = \beta_2 z_{42} + \beta_4 z_{43} + \xi z_{15} - (\lambda_2 + \Omega) z_{45}, \quad (40)$$

$$SR^+ \quad \dot{z}_{16} = \lambda_1 z_{14} + \lambda_2 z_{15} - (\nu + \xi + \Omega) z_{16}, \quad (41)$$

$$IR^+ \quad \dot{z}_{26} = \lambda_1 z_{24} + \lambda_2 z_{25} + \nu z_{16} - (\zeta + \omega\Omega) z_{26}, \quad (42)$$

$$RR^+ \quad \dot{z}_{36} = \lambda_1 z_{34} + \lambda_2 z_{35} + \zeta z_{26} - \Omega z_{36}, \quad (43)$$

$$VR^+ \quad \dot{z}_{46} = \lambda_1 z_{44} + \lambda_2 z_{45} + \xi z_{16} - \Omega z_{46}, \quad (44)$$

$$SR^- \quad \dot{z}_{17} = \lambda_1 z_{14} + \lambda_2 z_{15} - (\nu + \xi + \Omega) z_{17}, \quad (45)$$

$$IR^- \quad \dot{z}_{27} = \lambda_1 z_{24} + \lambda_2 z_{25} + \nu z_{17} - (\zeta + \omega\Omega) z_{27}, \quad (46)$$

$$RR^- \quad \dot{z}_{37} = \lambda_1 z_{34} + \lambda_2 z_{35} + \zeta z_{27} - \Omega z_{37}, \quad (47)$$

$$VR^- \quad \dot{z}_{47} = \lambda_1 z_{44} + \lambda_2 z_{45} + \xi z_{17} - \Omega z_{47}. \quad (48)$$

.1 Perturbed equations full

For each information class $j \in \{S, E, D, P, N, R^+, R^-\}$ define the time-dependent infection rate

$$\kappa_j(t) := \beta_0 \varphi_{\text{sus}}(\pi(t)) c_j(\pi(t)) I_{\text{eff}}(t),$$

where $\beta_0 > 0$ is the baseline transmission rate, φ_{sus} is the sentiment-dependent susceptibility modifier, $c_j(\pi)$ is the class-specific weight, and $I_{\text{eff}}(t)$ is the effective infectious density. The perturbed system for the densities $z_{ij}(t)$, ordered with viral states (S_v, I_v, R_v, V_v) and information states $(S, E, D, P, N, R^+, R^-)$, is then what we present on the next page.

$$SS \quad \dot{z}_{11} = B + \mu_1 z_{12} + \mu_2 z_{13} - (\alpha + \nu + \xi + \Omega) z_{11} - \kappa_S(t) z_{11}, \quad (49)$$

$$IS \quad \dot{z}_{21} = \mu_1 z_{22} + \mu_2 z_{23} + \nu z_{11} - (\alpha + \zeta + \omega\Omega) z_{21} + \kappa_S(t) z_{11}, \quad (50)$$

$$RS \quad \dot{z}_{31} = \mu_1 z_{32} + \mu_2 z_{33} + \zeta z_{21} - (\alpha + \Omega) z_{31}, \quad (51)$$

$$VS \quad \dot{z}_{41} = \mu_1 z_{42} + \mu_2 z_{43} + \xi z_{11} - (\alpha + \Omega) z_{41}, \quad (52)$$

$$SE \quad \dot{z}_{12} = \alpha z_{11} - (\nu + \xi + \mu_1 + \beta_1 + \beta_2 + \gamma + \Omega) z_{12} - \kappa_E(t) z_{12}, \quad (53)$$

$$IE \quad \dot{z}_{22} = \alpha z_{21} + \nu z_{12} - (\zeta + \mu_1 + \beta_1 + \beta_2 + \gamma + \omega\Omega) z_{22} + \kappa_E(t) z_{12}, \quad (54)$$

$$RE \quad \dot{z}_{32} = \alpha z_{31} + \zeta z_{22} - (\mu_1 + \beta_1 + \beta_2 + \gamma + \Omega) z_{32}, \quad (55)$$

$$VE \quad \dot{z}_{42} = \alpha z_{41} + \xi z_{12} - (\mu_1 + \beta_1 + \beta_2 + \gamma + \Omega) z_{42}, \quad (56)$$

$$SD \quad \dot{z}_{13} = \gamma z_{12} - (\nu + \xi + \mu_2 + \beta_3 + \beta_4 + \Omega) z_{13} - \kappa_D(t) z_{13}, \quad (57)$$

$$ID \quad \dot{z}_{23} = \gamma z_{22} + \nu z_{13} - (\zeta + \mu_2 + \beta_3 + \beta_4 + \omega\Omega) z_{23} + \kappa_D(t) z_{13}, \quad (58)$$

$$RD \quad \dot{z}_{33} = \gamma z_{32} + \zeta z_{23} - (\mu_2 + \beta_3 + \beta_4 + \Omega) z_{33}, \quad (59)$$

$$VD \quad \dot{z}_{43} = \gamma z_{42} + \xi z_{13} - (\mu_2 + \beta_3 + \beta_4 + \Omega) z_{43}, \quad (60)$$

$$SP \quad \dot{z}_{14} = \beta_1 z_{12} + \beta_3 z_{13} - (\nu + \xi + \lambda_1 + \Omega) z_{14} - \kappa_P(t) z_{14}, \quad (61)$$

$$IP \quad \dot{z}_{24} = \beta_1 z_{22} + \beta_3 z_{23} + \nu z_{14} - (\zeta + \lambda_1 + \omega\Omega) z_{24} + \kappa_P(t) z_{14}, \quad (62)$$

$$RP \quad \dot{z}_{34} = \beta_1 z_{32} + \beta_3 z_{33} + \zeta z_{24} - (\lambda_1 + \Omega) z_{34}, \quad (63)$$

$$VP \quad \dot{z}_{44} = \beta_1 z_{42} + \beta_3 z_{43} + \xi z_{14} - (\lambda_1 + \Omega) z_{44}, \quad (64)$$

$$SN \quad \dot{z}_{15} = \beta_2 z_{12} + \beta_4 z_{13} - (\nu + \xi + \lambda_2 + \Omega) z_{15} - \kappa_N(t) z_{15}, \quad (65)$$

$$IN \quad \dot{z}_{25} = \beta_2 z_{22} + \beta_4 z_{23} + \nu z_{15} - (\zeta + \lambda_2 + \omega\Omega) z_{25} + \kappa_N(t) z_{15}, \quad (66)$$

$$RN \quad \dot{z}_{35} = \beta_2 z_{32} + \beta_4 z_{33} + \zeta z_{25} - (\lambda_2 + \Omega) z_{35}, \quad (67)$$

$$VN \quad \dot{z}_{45} = \beta_2 z_{42} + \beta_4 z_{43} + \xi z_{15} - (\lambda_2 + \Omega) z_{45}, \quad (68)$$

$$SR^+ \quad \dot{z}_{16} = \lambda_1 z_{14} + \lambda_2 z_{15} - (\nu + \xi + \Omega) z_{16} - \kappa_{R^+}(t) z_{16}, \quad (69)$$

$$IR^+ \quad \dot{z}_{26} = \lambda_1 z_{24} + \lambda_2 z_{25} + \nu z_{16} - (\zeta + \omega\Omega) z_{26} + \kappa_{R^+}(t) z_{16}, \quad (70)$$

$$RR^+ \quad \dot{z}_{36} = \lambda_1 z_{34} + \lambda_2 z_{35} + \zeta z_{26} - \Omega z_{36}, \quad (71)$$

$$VR^+ \quad \dot{z}_{46} = \lambda_1 z_{44} + \lambda_2 z_{45} + \xi z_{16} - \Omega z_{46}, \quad (72)$$

$$SR^- \quad \dot{z}_{17} = \lambda_1 z_{14} + \lambda_2 z_{15} - (\nu + \xi + \Omega) z_{17} - \kappa_{R^-}(t) z_{17}, \quad (73)$$

$$IR^- \quad \dot{z}_{27} = \lambda_1 z_{24} + \lambda_2 z_{25} + \nu z_{17} - (\zeta + \omega\Omega) z_{27} + \kappa_{R^-}(t) z_{17}, \quad (74)$$

$$RR^- \quad \dot{z}_{37} = \lambda_1 z_{34} + \lambda_2 z_{35} + \zeta z_{27} - \Omega z_{37}, \quad (75)$$

$$VR^- \quad \dot{z}_{47} = \lambda_1 z_{44} + \lambda_2 z_{45} + \xi z_{17} - \Omega z_{47}. \quad (76)$$