Coupling Environmental Exposure and Human Mobility in an SEIR Metapopulation Model of Influenza Transmission

Application to Influenza Dynamics in Portugal

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Introduction

Respiratory infections such as influenza and RSV remain a **major public health burden** and exhibit **strong seasonal patterns** [1].

In **temperate regions**, incidence rises in autumn, peaks during winter, and declines in spring, while **distinct seasonal patterns** are observed in **tropical climates** [2, 3, 4].

Three main mechanisms have been proposed to explain viral seasonality:

- **Host Resistance**: Climatic conditions weaken the body's ability to fight infection – higher susceptibility (e.g., reduced sun exposure leading to low Vitamin D levels).
- **Virus Survival**: Temperature and humidity directly affect the structural integrity and environmental survival of the virus [5, 6].
- **Behavioural Changes**: Changes in human mobility and social mixing (e.g., indoor congregation, school terms) facilitate transmission. Human contact patterns and mobility are key determinants of transmission dynamics and spatial spread [7].

Overall, environmental factors act as modulators of transmission intensity, interacting with **contact patterns and mobility** rather than acting as direct causes [8].

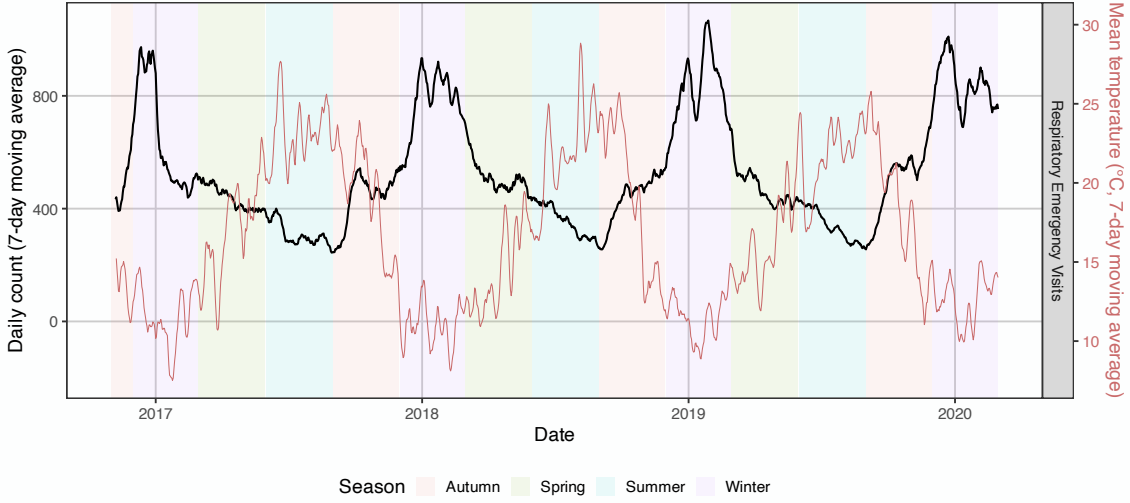


Figure 1: 7-day moving averages of influenza consultations and respiratory urgencies in ARS Lisboa e Vale do Tejo. Dashed line shows daily mean temperature. Coloured backgrounds indicate seasons, highlighting seasonal trends and environmental influence on healthcare demand.

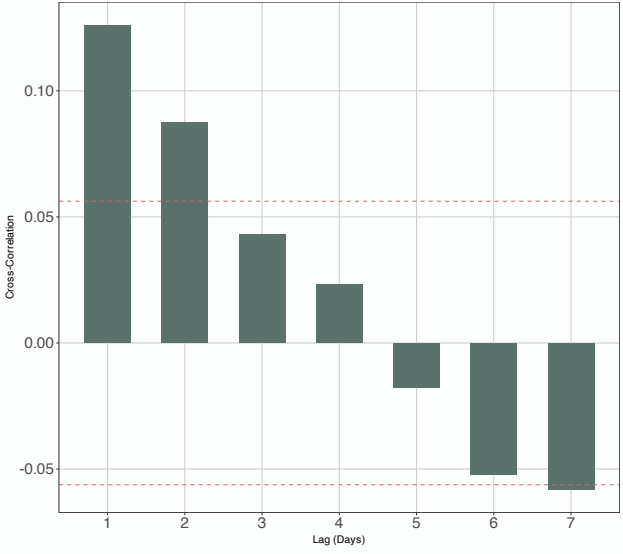


Figure 2: Cross-correlation showing delayed temperature effect.

Objectives

Aim

Understand how temperature-driven changes in viral efficiency and human contacts shape influenza seasonality, including spatial spread through mobility.

Objectives

1. Define a coupled transmission model where temperature modulates both virus transmissibility and contact rates.
2. Compare alternative temperature-dependent functional forms in the coupled SEIR model.
3. Extend the model to a metapopulation framework and apply it to a Regional Health Administration in Portugal.

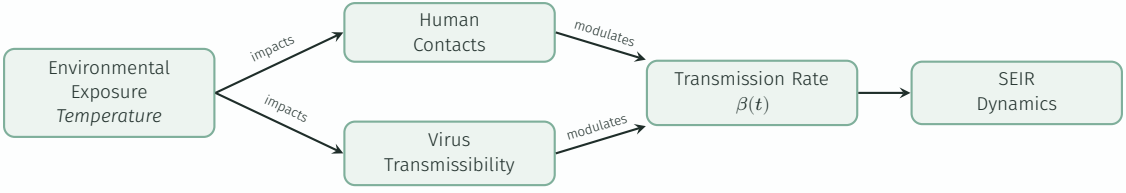


Figure 3: Impact of environmental conditions and mobility on transmission.

Coupled SEIR Model

Model Description

We consider a SEIR-type model,

- S: susceptible individuals
- E: exposed (infected, not yet infectious)
- I: infectious
- R: removed individuals
- $T(t)$: Temperature over time t

- Infection: $S \to E$ at rate $\lambda\left(T(t)\right) S$.
- Progression: $E \to I$ with probability $1-p$ at rate σ.
- Recovery: $I \to R$ at rate γ.

$$
\begin{aligned}
S' &= -\lambda\left(T(t)\right)\, S, \\
E' &= \lambda\left(T(t)\right)\, S - \sigma E, \\
I' &= \sigma E - \gamma I, \\
R' &= \gamma I
\end{aligned}
$$

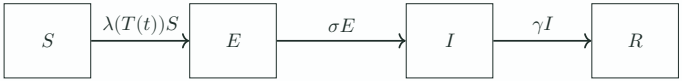


Figure 4: Schematic representation of the SEIR compartmental

Force of Infection

Key Idea

Temperature acts as a *modulator* of transmission by influencing **how often people interact** and **how efficiently the virus spreads**.

$$\lambda\left(T(t)\right) = \beta\left(T(t)\right)\,\frac{I(t)}{N(t)}$$

Two complementary pathways

- **Virus Transmissibility** Cold air enhances viral survival and weakens nasal immune defences.
- **Human Contacts** Lower temperatures shift contacts indoors, increasing proximity and reducing ventilation.

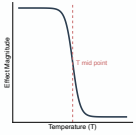
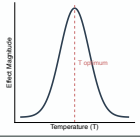
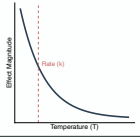
The temperature-dependent transmission rate $\beta(T(t))$ is defined as the product of the baseline probability of transmission per contact β_{base} the virus transmissibility $V(T(t))$ and the contact rate $C(T(t))$:

$$\beta(T(t)) = \beta_{\text{base}}\, V(T(t))\, C(T(t))$$

Key question

What functional form should we choose for the temperature-dependent transmission for $E(\cdot)$ and $V(\cdot)$?

Table 1: Comparative analysis of functional forms. Three functions (Sigmoid, Gaussian, Exponential) characterizing temperature-dependent contact rates and virus transmissibility and their practical interpretations in an epidemiological context. Plots are *schematic and scale-agnostic*, designed to highlight shape differences.

Type	Shape	Function Form	Contacts $C(T)$	Viral Transmissibility $V(T)$
Sigmoid Saturation		$y = y_{\min} + \frac{\Delta y}{1+e^{k(T-T_{mid})}}$	Contacts rise as temperature drops, but level off once most activity is indoors.	Virus remains stable in cold conditions, then loses viability beyond a threshold temperature.
Gaussian Optimum Interval		$y = y_{\min} + \Delta y\, e^{-\frac{(T-T_{opt})^2}{2\sigma^2}}$	Contacts peak at a preferred temperature or event, and decrease if conditions are too cold or too warm.	Virus performs best at an optimal temperature, with reduced survival at both lower and higher temperatures.
Exponential Steady Decay		$y = y_{\min} + \Delta y\, e^{-k(T-T_{ref})}$	Contacts change gradually with temperature, without sharp behavioural transitions.	Viral survival decreases steadily as temperature increases.

Note: $\Delta y = y_{\max} - y_{\min}$

$$\beta(T(t)) = \beta_{\text{base}}\, E(T(t))\, C(T(t))$$

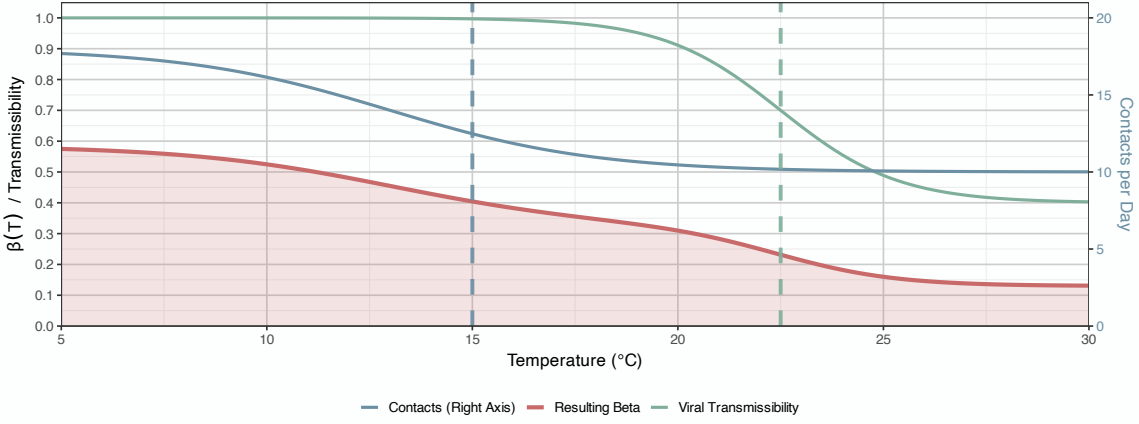


Figure 5: Temperature-dependent transmission mechanism. Viral transmissibility (green, left axis) and human contact intensity (blue, right axis) respond differently to temperature. Their interaction produces the resulting transmission rate $\beta(T)$ (without the scaling parameter β_{base} optimized, $\beta_{\text{base}} = 1$) (red, solid), with the shaded area. Vertical dotted lines indicate characteristic temperature thresholds for biological stability and behavioural change.

Application of the Coupled Model

Model Setup

To illustrate the **SEIR Coupled Model**, we explore combinations of constant vs. sigmoidal formulations for both contact rate and virus transmissibility functions.

Parameters are calibrated to match **seasonal influenza** characteristics reported in the literature.

- **Latent Period** $1/\sigma \approx 3$ **days**: Mean incubation period of seasonal influenza is approximately 2–3 days [9].
- **Infectious Period** $1/\gamma \approx 4$ **days**: Infectiousness peaks shortly after symptom onset and typically resolves within 5–7 days [10].
- **Viral Transmissibility** $V(T)$: Temperature-dependent transmissibility, with a pronounced decline above 20–25 °C due to reduced viral survival and transmission [13].
- **Contact Rate** $C_{\mathbf{min}} = 10,\ C_{\mathbf{max}} = 18$ **contacts/day**: Behavioural variation in daily contacts, centred on European survey estimates; contacts begin to shift as temperatures rise into the 15–20 °C range [14, 15].
- **Baseline Transmission Probability** $\beta_{\text{base}} = 4\%$: Calibrated to reproduce $R_t \approx 2$ under peak winter conditions with temperature effects [16], and $R_t \approx 1.5$ in scenarios without seasonal dependence [17].
- **Initial conditions considered**: $S_0 = 9\,750$, $E_0 = 100$, $I_0 = 150$, $I_{h,0} = 0$, $R_0 = 0$

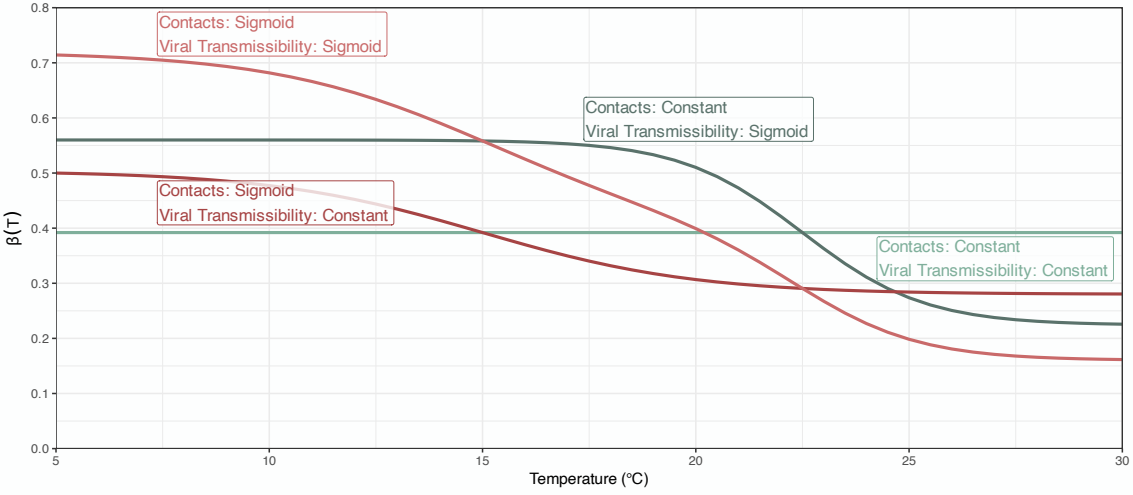


Figure 6: Temperature dependence of the transmission rate $\beta(T)$. The figure displays the calculated transmission rate β as a function of temperature (5–30°C) for four scenarios. Labels indicate the combination of functional forms used for the contact rate $C(T)$ and virus transmissibility $V(T)$ (Constant vs. Sigmoidal).

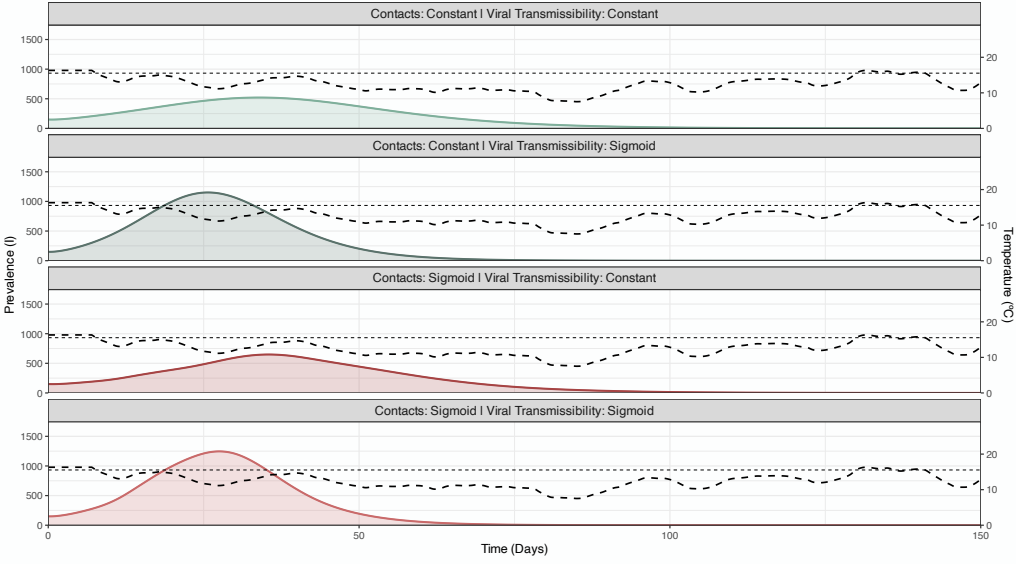


Figure 7: Epidemic prevalence (infectious compartment I) over time for four coupling scenarios. Dashed black lines represent the temperature time series (scaled to the secondary axis which displays temperature in °C) and its mean value.

Extending to a Coupled SEIR Metapopulation Model

Why a metapopulation model?

Seasonal influenza does not evolve in a single, well-mixed population:

- **Spatial temperature heterogeneity** drives regional differences in transmissibility.
- **Human mobility** connects regions and enables spatial spread.

The population is divided into **interconnected patches**, each following local SEIR dynamics with **temperature-dependent transmission**. Mobility links patches, allowing infections to travel.

Why this coupling is useful:

- It introduces **environmental heterogeneity**, allowing contact rates to adapt to local climatic conditions (e.g., climate-specific behavioural responses).
- It enables **patch-specific mobility or contact interventions** during predefined time periods (e.g., non-pharmaceutical interventions affecting mixing or movement).
- It allows **time-varying transmissibility**, such as the introduction of new viral strains with different transmission potential.

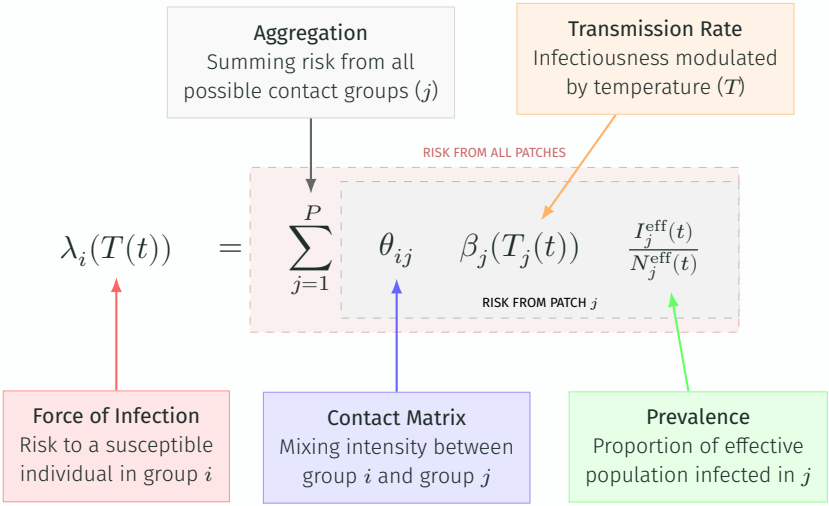


Figure 8: Schematic decomposition of the force of infection, $\lambda_i(T(t))$. The equation aggregates the risk from all groups j, weighted by the contact matrix θ_{ij} and the temperature-dependent transmission rate β_j.

Force of Infection

Residents of patch i accumulate infection risk across all patches they visit.

$$\lambda_i(T(t)) = \sum_{j=1}^{P} \theta_{ij}\, \beta_j(T_j(t))\, \frac{I_j^{\text{eff}}(t)}{N_j^{\text{eff}}(t)}$$

- $\beta_j(T)$ is the transmission as function of temperature T in patch j.
- θ_{ij} is the fraction of time a resident of patch i spends in patch j, $\sum_{j=1}^{P} \theta_{ij} = 1\ \forall i$ (time conservation).
- The **effective total population** in patch j is the sum of all visitors,

$$N_j^{\text{eff}}(t) = \sum_{k=1}^{P} \theta_{kj}\, (S_k(t) + E_k(t) + I_k(t) + R_k(t))$$

- The **effective infectious population** in patch j is the sum of infectious visitors,

$$I_j^{\text{eff}}(t) = \sum_{k=1}^{P} \theta_{kj} I_k(t)$$

Spatial structure and population.
Each patch represents a municipality within the Regional Health Administration (ARS) Lisboa and Tagus Valley (plus an "Outside" node for external mobility). Municipal populations [18] define patch sizes N_i and initial conditions:

$$S_i(0) = N_i, \quad E_i(0) = I_i(0) = R_i(0) = 0. \qquad \sum_{i \neq \text{Outside}} N_i = 3\,683\,392 \qquad \sum_i N_i = 5\,8211\,53$$

Temperature coupling.
Patch-specific temperature time series $T_i(t)$ drive local transmissibility and contact rates through $\beta(T_i(t))$.

Mobility network.
Inter-municipal mobility is derived from an Origin–Destination matrix of **pendular movements** [19], normalized to represent the fraction of time residents of patch i spend in patch j.

Epidemic seeding.
Infections are seeded in the K most populous municipalities (high-connectivity hubs), with 0.1% of the local population split evenly between E_i and I_i. All remaining parameters follow the seasonal influenza specification defined previously.

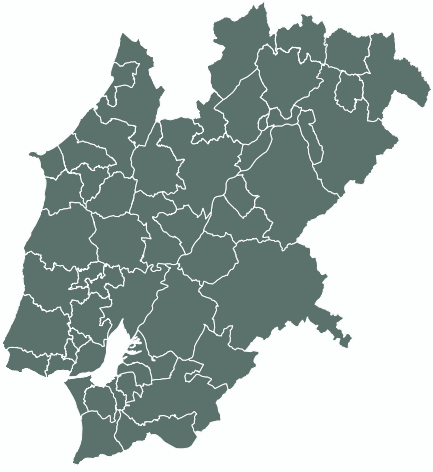


Figure 9: Geographical map of the Regional Health Region (ARS) Lisboa.

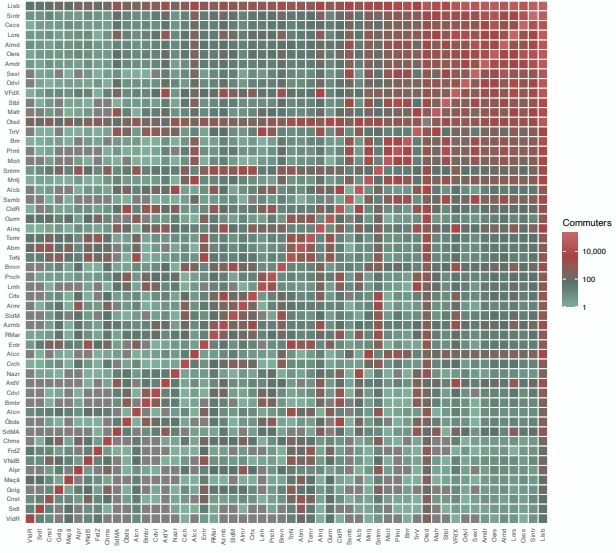


Figure 10: Log-scaled origin-destination flows within Regional Health Region (ARS) Lisboa.

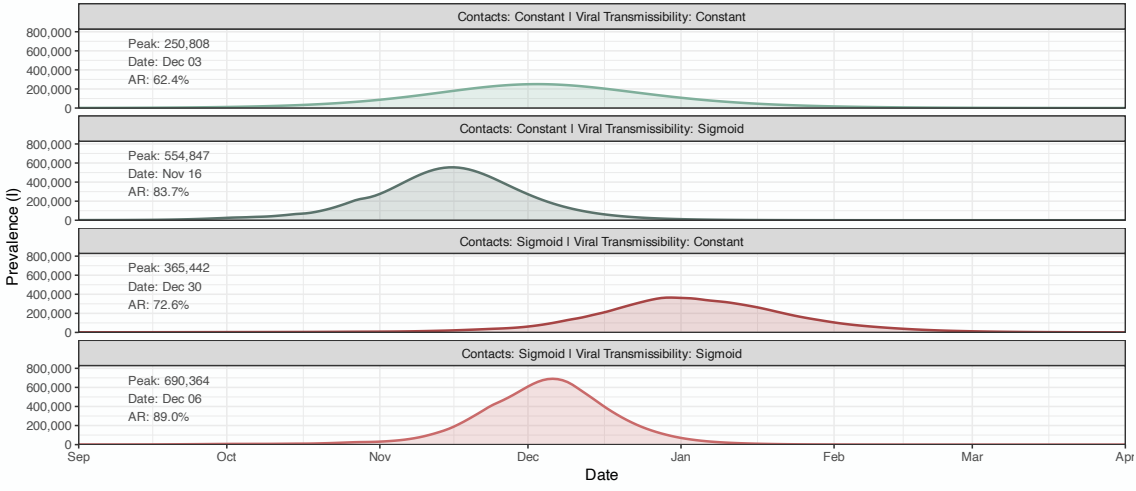


Figure 11: Simulated infection prevalence (I). The panels display four scenarios varying by contact patterns seasonality (Contacts) and Viral Transmissibility. The coloured areas represent the simulated prevalence of infectious individuals (I). Text annotations highlight the peak prevalence, the date of the peak, and the cumulative attack rate (AR) for each scenario. Environmental coupling shifted the peak timing, the magnitude of the onset and modulated the corresponding decline.

Discussion

Discussion

Constant Parameters (Null Model)

- **Driver:** Epidemic trajectory determined solely by R_0 and initial seeding.
- **Outcome:** Peak occurs too early or is overly broad, driven purely by the depletion of susceptibles.
- **Mismatch:** Fails to capture the specific winter surge observed in the data.

Environmental Coupling Model

- **Driver:** Transmission probability $\beta(t)$ is modulated by environmental forcing.
- **Mechanism:** Growth is suppressed during "unfavourable" months, delaying the exponential phase.
- **Result:** The peak is forced into the window of maximum environmental receptivity (Winter).

Key Advantages

- This design allows **time-varying contact rates**, capturing the effects of non-pharmaceutical interventions (e.g., lockdowns) independently of environmental factors.
- **Flexible virus transmissibility parameters** enable the introduction of multiple strains with **distinct temperature sensitivities**.

Thank you for your attention!

Any questions?

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Contacts

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Scan for the GitHub repository with slides, data and references.

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