

Article

A Two-City, Two-Group SIR Model with Heterogeneous Risk-Perception Feedback and Group-Specific Mobility

Yerikkyzy Arailym^{1*}¹School of Information Technology and Applied Mathematics, SDU University, Kazakhstan

DOI: 10.47344/mqecf280

Abstract

We formulate and analyze a deterministic two-city, two-group susceptible–infected–recovered (SIR) model with heterogeneous behavioral adaptation and group-specific mobility. The model combines two mechanisms: (i) group-dependent contact reduction in response to local infection burden and (ii) symmetric inter-city diffusion with group-dependent mobility rates. Using a next-generation matrix construction, we derive the invasion threshold and compute R_0 under mobility coupling. Numerical experiments identify three robust dynamical regimes: weak coupling with delayed spillover, intermediate coupling with partial synchronization, and strong coupling with near-synchronous outbreaks. Parameter sweeps over high-contact-group mobility and risk sensitivity show nonlinear mobility–behavior interactions affecting peak burden and final size. Across scenarios, interventions targeted at the high-contact group (increased risk sensitivity and/or reduced mobility) yield the largest burden reductions, and combined interventions dominate single-mechanism policies.

Keywords: SIR model, metapopulation, mobility, behavioral feedback, risk perception, heterogeneous mixing, next-generation matrix, epidemic synchronization

I. INTRODUCTION

The spread of infectious diseases across connected urban centers is shaped by two broad drivers: (i) movement of individuals between locations and (ii) behavioral adaptation in response to perceived risk. The classical deterministic compartmental models usually make assumptions that include constant contact rates as well as homogeneous mixing, which may mask the essential processes that determine when sync-ups take place, when spillovers take place, as well as when targeted interventions are effective [1], [14]. In reality, populations are not homogeneous because some subgroups tend to have greater contact rates as a result of their occupation or social behavior. Additionally, individuals tend to reduce their contacts as local case numbers increase, as has been seen in various epidemics, as described by prevalence-dependent contact functions [8]–[10].

*Corresponding author: arailym.yerikkyzy@mail.ru

Email: arailym.yerikkyzy@mail.ru ORCID: 0009-0009-9675-0044

Metapopulation models have been used extensively in understanding the pattern of spread of diseases over areas and their entry into new areas [3], [5], [6], [12]. However, most approaches have been based on the assumption that mobility is uniform across all population groups. This is a limitation, as in practice, mobility patterns may differ between groups. For example, in the case of high-contact groups, mobility is more for work-related activities, and in the case of cautious groups, mobility is less. In addition, behavioral feedback is typically assumed in an aggregate manner. However, there are considerable variations in the perception of risk and compliance in different groups [4], [15], [16], [18].

In this article, a tractable two-city, two-group SIR model is proposed that combines (a) heterogeneous risk perception feedback and (b) group-specific mobility. Compared to models that include either only behavioral effects or only mobility effects, the proposed model combines both effects in a unified analytical framework, which allows for the identification of both threshold and synchronization effects. The model is also designed to be simple so that next-generation matrix analysis and simulations are transparent and efficient.

A. Contributions

Our main contributions can be summarized as follows:

- We propose a model for two cities with SIR dynamics and two types of behavior with different basic contact rates, sensitivity to risk, and mobility.
- We propose a behavior feedback mechanism in which group-specific contact rates decrease exponentially with respect to local infection pressure.
- We derive the invasion threshold using the next-generation matrix and calculate R_0 with mobility coupling.
- We identify qualitative regimes of mobility (weak, intermediate, and fast) and their effects on the timing of spillovers and their synchronization.
- We comparatively evaluate targeted interventions for the high-contact group using risk communication (to enhance sensitivity) and/or reducing mobility.

II. MODEL FORMULATION

We focus on two cities, $X \in \{A, B\}$, and two population groups, $g \in \{1, 2\}$. Each of these cities and population groups is further divided into susceptible $S_{Xg}(t)$, infected $I_{Xg}(t)$, and recovered $R_{Xg}(t)$ classes. Let

$$N_X(t) = \sum_{g=1}^2 (S_{Xg}(t) + I_{Xg}(t) + R_{Xg}(t)) \quad (1)$$

denote the total population in city X . We assume recovery at rate $\gamma > 0$ and transmission parameter $\beta > 0$.

A. Model Assumptions

For ease of explanation, the basic assumptions of the model are:

- **Closed two-city system:** there are no births, deaths, and external migrations over the course of the model.
- **Group structure:** there are two behavioral groups that only differ by their baseline contact rates (c_g^0), risk response (α_g), and mobility (m_g).
- **Local risk response:** the effective contact rates are determined by the local infection burden.
- **Within-city mixing:** transmission is city-level mass-action with shared hazard $\lambda_X(t)$ across groups.
- **Symmetric mobility:** movement between cities is bidirectional and group specific, preserving total population.
- **Constant recovery:** all infected individuals recover at rate γ , with no reinfection (SIR immunity).
- **Deterministic dynamics:** stochastic fadeout and random importation events are not modeled.

B. Notation and baseline parameters

Table I summarizes key model variables and parameters used throughout the analysis. All simulations use the baseline values listed above unless otherwise stated. These parameters fully define all numerical experiments reported in the paper.

TABLE I
MODEL PARAMETERS AND BASELINE VALUES USED IN ALL SIMULATIONS

Symbol	Meaning	Baseline value / range
S_{Xg}, I_{Xg}, R_{Xg}	Susceptible, infected, recovered in city X , group g	State variables
N_X	Total population in city X	$N_A = N_B = 1200$
β	Transmission coefficient	$\beta = 0.24$
γ	Recovery rate	$\gamma = 0.09$
c_g^0	Baseline contact rate (group g)	$c_1^0 = 0.9, c_2^0 = 2.0$
α_g	Risk sensitivity (group g)	$\alpha_1 = 8.0, \alpha_2 = 2.2$
m_g	Inter-city mobility rate (group g)	$m_1 = 0.05, m_2 \in [0.005, 0.35]$
λ_X	Force of infection in city X	see Eq. (3)
R_0	Basic reproduction number	$\rho(FV^{-1})$
Initial conditions		
Infection seeding		$I_{A2}(0) = 8$, all other infected = 0
Recovered		$R_{Xg}(0) = 0$
Susceptible		$S_{Xg}(0) = N_{Xg} - I_{Xg}(0)$

C. Risk-perception feedback on contact rates

Behavior is incorporated through group-specific effective contact rates in each city. Let c_g^0 be the baseline contact rate for group g in the absence of perceived risk, and let $\alpha_g \geq 0$ be the risk sensitivity parameter. We model contact reduction as a decreasing function of the local infection prevalence $I_X(t)/N_X(t)$, where $I_X(t) = I_{X1}(t) + I_{X2}(t)$:

$$c_g^X(t) = c_g^0 \exp\left(-\alpha_g \frac{I_X(t)}{N_X(t)}\right). \quad (2)$$

This choice captures rapid behavioral change under increasing local burden and yields a smooth contact function bounded above by c_g^0 . Larger α_g implies a stronger behavioral response.

D. Force of infection

Within each city, infections are generated by contacts with infected individuals from both groups. We define the force of infection in city X as

$$\lambda_X(t) = \beta \frac{c_1^X(t)I_{X1}(t) + c_2^X(t)I_{X2}(t)}{N_X(t)}. \quad (3)$$

In the present formulation, both groups experience the same hazard $\lambda_X(t)$ given the shared infectious pool and city-level mixing intensity.

E. Group-specific mobility

Mobility is modeled as symmetric exchange between cities with group-dependent rates $m_g \geq 0$. Individuals in group g move from A to B and from B to A at rate m_g , preserving total population across the two-city system. This structure follows standard multi-city compartmental constructions while allowing explicit group heterogeneity in movement [2].

F. Dynamical system

For each group g , the SIR dynamics with mobility are

$$\frac{dS_{Ag}}{dt} = -\lambda_A S_{Ag} + m_g S_{Bg} - m_g S_{Ag}, \quad (4)$$

$$\frac{dI_{Ag}}{dt} = \lambda_A S_{Ag} - \gamma I_{Ag} + m_g I_{Bg} - m_g I_{Ag}, \quad (5)$$

$$\frac{dR_{Ag}}{dt} = \gamma I_{Ag} + m_g R_{Bg} - m_g R_{Ag}, \quad (6)$$

and similarly for city B ,

$$\frac{dS_{Bg}}{dt} = -\lambda_B S_{Bg} + m_g S_{Ag} - m_g S_{Bg}, \quad (7)$$

$$\frac{dI_{Bg}}{dt} = \lambda_B S_{Bg} - \gamma I_{Bg} + m_g I_{Ag} - m_g I_{Bg}, \quad (8)$$

$$\frac{dR_{Bg}}{dt} = \gamma I_{Bg} + m_g R_{Ag} - m_g R_{Bg}, \quad (9)$$

with $\lambda_X(t)$ given by (3) and $c_g^X(t)$ given by (2).

G. Disease-free equilibrium

At the disease-free equilibrium (DFE), $I_{Xg} = 0$ and $R_{Xg} = 0$, hence $S_{Xg} = N_{Xg}^0$ and $c_g^X = c_g^0$. This equilibrium is used to derive the invasion threshold.

H. Well-posedness and invariant region

For each group $g \in \{1, 2\}$, define the group total across both cities:

$$M_g(t) = S_{Ag}(t) + I_{Ag}(t) + R_{Ag}(t) + S_{Bg}(t) + I_{Bg}(t) + R_{Bg}(t).$$

Summing the corresponding six equations shows

$$\frac{dM_g}{dt} = 0, \quad g \in \{1, 2\},$$

so $M_g(t) = M_g(0)$ for all $t \geq 0$. Hence total population is conserved: $M_1(t) + M_2(t) = M_1(0) + M_2(0)$.

Proposition 1 (Positivity and forward invariance). Assume nonnegative initial conditions and parameters $\beta, \gamma, m_g, \alpha_g \geq 0$. Then all state variables remain nonnegative for all $t \geq 0$, and trajectories remain in the compact positively invariant set

$$\Omega = \left\{ (S, I, R) \in \mathbb{R}_{\geq 0}^{12} : \sum_{X \in \{A, B\}} (S_{Xg} + I_{Xg} + R_{Xg}) = M_g(0), \quad g = 1, 2 \right\}.$$

Proof. The right-hand side is locally Lipschitz because the contact feedback (2) and force of infection (3) are smooth for $N_X(t) > 0$; conservation of M_g guarantees N_X remains bounded and positive for epidemiologically meaningful initial conditions. On each boundary hyperplane (e.g., $S_{Ag} = 0$), the corresponding derivative is nonnegative:

$$\dot{S}_{Ag}|_{S_{Ag}=0} = m_g S_{Bg} \geq 0, \quad \dot{I}_{Ag}|_{I_{Ag}=0} = \lambda_A S_{Ag} + m_g I_{Bg} \geq 0, \quad \dot{R}_{Ag}|_{R_{Ag}=0} = \gamma I_{Ag} + m_g R_{Bg} \geq 0,$$

and analogously for city B . Therefore trajectories cannot cross into negative values. Conservation of M_g then implies boundedness in Ω .

III. THRESHOLD ANALYSIS AND BASIC REPRODUCTION NUMBER

We compute the basic reproduction number R_0 using a next-generation matrix approach [7], [17]. Let the infected-state vector be ordered as

$$\mathbf{i} = (I_{A1}, I_{A2}, I_{B1}, I_{B2})^\top. \quad (10)$$

Linearizing the system around the DFE yields

$$\dot{\mathbf{i}} = (F - V)\mathbf{i}, \quad (11)$$

where F captures new infections and V captures transitions out of infection (recovery and mobility).

A. New infection matrix (F)

At the DFE, $N_X = N_X^0$ and $c_g^X = c_g^0$. The force of infection in city A becomes

$$\lambda_A = \beta \frac{c_1^0 I_{A1} + c_2^0 I_{A2}}{N_A^0}. \quad (12)$$

Thus, new infections entering group g in city A are

$$\lambda_A S_{Ag} = \beta \frac{N_{Ag}^0}{N_A^0} (c_1^0 I_{A1} + c_2^0 I_{A2}). \quad (13)$$

This gives the block

$$F_A = \beta \begin{pmatrix} c_1^0 \frac{N_{A1}^0}{N_A^0} & c_2^0 \frac{N_{A1}^0}{N_A^0} \\ c_1^0 \frac{N_{A2}^0}{N_A^0} & c_2^0 \frac{N_{A2}^0}{N_A^0} \end{pmatrix}. \quad (14)$$

Similarly for city B ,

$$F_B = \beta \begin{pmatrix} c_1^0 \frac{N_{B1}^0}{N_B^0} & c_2^0 \frac{N_{B1}^0}{N_B^0} \\ c_1^0 \frac{N_{B2}^0}{N_B^0} & c_2^0 \frac{N_{B2}^0}{N_B^0} \end{pmatrix}. \quad (15)$$

The full infection matrix is block diagonal:

$$F = \begin{pmatrix} F_A & 0 \\ 0 & F_B \end{pmatrix}. \quad (16)$$

B. Transition matrix (V)

Transitions out of infected states occur via recovery and mobility. In the ordering $(A1, A2, B1, B2)$, the matrix is

$$V = \begin{pmatrix} \gamma + m_1 & 0 & -m_1 & 0 \\ 0 & \gamma + m_2 & 0 & -m_2 \\ -m_1 & 0 & \gamma + m_1 & 0 \\ 0 & -m_2 & 0 & \gamma + m_2 \end{pmatrix}. \quad (17)$$

C. Next-generation matrix and basic reproduction number

We now construct the next-generation matrix using F and V defined above. It is convenient to define

$$d_g = \gamma(\gamma + 2m_g), \quad g \in \{1, 2\}. \quad (18)$$

Then

$$V^{-1} = \begin{pmatrix} \frac{\gamma+m_1}{d_1} & 0 & \frac{m_1}{d_1} & 0 \\ 0 & \frac{\gamma+m_2}{d_2} & 0 & \frac{m_2}{d_2} \\ \frac{m_1}{d_1} & 0 & \frac{\gamma+m_1}{d_1} & 0 \\ 0 & \frac{m_2}{d_2} & 0 & \frac{\gamma+m_2}{d_2} \end{pmatrix}. \quad (19)$$

To display the coupling structure more transparently, define

$$D_+ = \text{diag} \left(\frac{\gamma + m_1}{d_1}, \frac{\gamma + m_2}{d_2} \right), \quad D_- = \text{diag} \left(\frac{m_1}{d_1}, \frac{m_2}{d_2} \right). \quad (20)$$

Hence,

$$V^{-1} = \begin{pmatrix} D_+ & D_- \\ D_- & D_+ \end{pmatrix}, \quad (21)$$

and the next-generation matrix becomes

$$K = FV^{-1} = \begin{pmatrix} F_A D_+ & F_A D_- \\ F_B D_- & F_B D_+ \end{pmatrix}. \quad (22)$$

The basic reproduction number is defined as the spectral radius of K :

$$R_0 = \rho(K). \quad (23)$$

The diagonal blocks represent within-city transmission, while the off-diagonal blocks capture cross-city infections induced by mobility. Thus, R_0 incorporates both heterogeneous group transmission and spatial coupling at invasion.

Theorem 1 (Local threshold characterization at the disease-free equilibrium). Consider the infection subsystem linearized at the DFE. The DFE is locally asymptotically stable if $R_0 < 1$ and is unstable if $R_0 > 1$.

Proof. The linearization of the infection system at the DFE is in standard next-generation form with $F \geq 0$ and a nonsingular M-matrix V for $\gamma > 0$ and $m_g \geq 0$. By the next-generation theorem, the sign of the dominant eigenvalue of $(F - V)$ is determined by whether $\rho(FV^{-1})$ is below or above unity. Therefore the threshold is exactly $R_0 = 1$ [7], [17].

D. Weak-mobility approximation (city-specific)

In the weak-mobility limit, each city behaves approximately as an uncoupled two-group SIR system at invasion. A convenient city-specific approximation is

$$R_0^{(X)} \approx \frac{\beta}{\gamma} \left(c_1^0 \frac{N_{X1}^0}{N_X^0} + c_2^0 \frac{N_{X2}^0}{N_X^0} \right), \quad X \in \{A, B\}. \quad (24)$$

This expression highlights how group composition and baseline contact rates shape local invasion thresholds and is consistent with threshold analyses for structured epidemic systems [19].

IV. NUMERICAL METHODS AND OUTCOME METRICS

All simulations use the baseline parameter values and initial conditions listed in Table I unless otherwise stated. The nonlinear system is solved using an explicit adaptive Runge–Kutta method (RK45). Initial conditions consist of susceptible populations equal to the DFE totals minus an initial infection seed, with all recovered states initialized at zero. A typical seeding scenario introduces a small number of infected individuals into the high-contact group in city A. All remaining compartments are initialized consistently with these assumptions.

A. Reproducibility settings

To support reproducibility, simulations are run on a fixed time horizon $[0, T]$ with a uniform reporting grid for all scenarios. Unless otherwise stated, we use deterministic initial conditions with a single-city seed in $(A, 2)$ and evaluate outcomes from the same state trajectories used for plotting. Parameter sweeps vary one or two policy-relevant parameters at a time (e.g., m_2 , α_2) while holding the remaining parameters at baseline values listed in Table I. This is the same process used for all sweep points: integration, metric extraction, and figure generation. We evaluate epidemic outcomes using the following quantitative metrics:

- Peak burden: $\max_t I_X(t)$ where $I_X(t) = I_{X1}(t) + I_{X2}(t)$.
- Time to peak: $t_X^* = \arg \max_t I_X(t)$.
- Final size (cumulative attack rate proxy): $R_X(\infty) = \lim_{t \rightarrow \infty} (R_{X1}(t) + R_{X2}(t))$.
- Spillover delay: $\Delta t = t_B^* - t_A^*$.
- Peak ratio: $\max_t I_B(t) / \max_t I_A(t)$.
- Synchronization index: correlation $\text{corr}(I_A(t), I_B(t))$ computed on the simulated time grid.

V. RESULTS

The results are organized around three main aspects: (i) qualitative mobility regimes and their effect on epidemic synchronization, (ii) the interaction between mobility and behavioral response, and (iii) the effectiveness of targeted interventions. Particular emphasis is placed on identifying non-obvious behaviors that arise from the coupling of mobility and adaptive contact reduction.

A. Mobility regimes: spillover and synchronization

Figure 1 illustrates representative epidemic trajectories under low, intermediate, and high mobility regimes. These regimes reveal a clear transition in the temporal relationship between outbreaks in the two cities.

In the low-mobility regime, the epidemic originates in city A and spreads to city B with a noticeable delay. This delayed spillover creates a temporal separation between epidemic peaks, resulting in low synchronization. Such a regime effectively provides a window for intervention in the initially unaffected city.

At intermediate mobility levels, the delay between outbreaks decreases, and the epidemic peaks begin to overlap. This leads to partial synchronization, where the correlation between epidemic curves increases but does not reach unity. Importantly, this regime represents a transitional phase in which both local dynamics and spatial coupling influence the system.

In the high-mobility regime, the system behaves similarly to a single well-mixed population. Epidemic peaks in both cities occur almost simultaneously, and synchronization is close to complete. In this case, spatial structure loses its mitigating effect, and interventions must be coordinated across locations.

Figure 1: Epidemic Trajectories Under Different Mobility Regimes

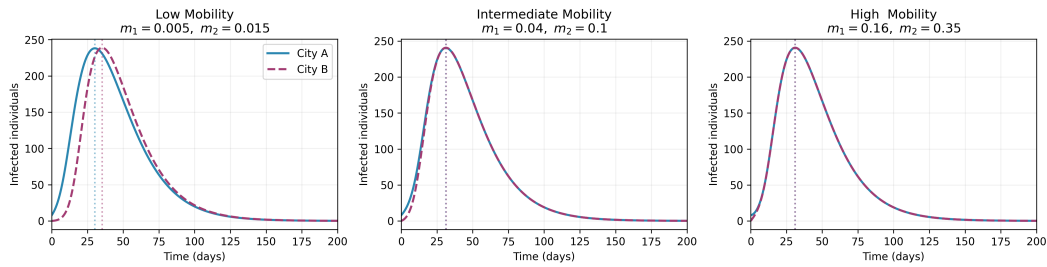


Fig. 1. Representative epidemic trajectories under three mobility regimes. Total infected individuals $I_A(t)$ and $I_B(t)$ are shown under low, intermediate, and high mobility rates. Vertical lines denote peak times. Low mobility yields delayed spillover; high mobility yields near-synchronous outbreaks.

B. Parameter landscape: nonlinear mobility–behavior interaction

We now examine how epidemic outcomes vary jointly as a function of the mobility of the high-contact group (m_2) and their risk sensitivity (α_2). Figure 2 presents heatmaps of peak infection levels in both cities across this parameter space.

Two general trends are observed. First, increasing α_2 consistently reduces the epidemic peak, reflecting stronger behavioral adaptation and contact reduction in response to local infection burden. Second, increasing m_2 enhances inter-city coupling, leading to faster spillover and increased transmission in the non-seeded city.

Beyond these expected effects, a key and non-obvious result emerges: the interaction between mobility and behavioral response is strongly nonlinear. In particular, under intermediate mobility levels, relatively small increases in risk sensitivity (α_2) can produce disproportionately large reductions in peak infection. This threshold-like behavior suggests that behavioral interventions may be especially effective when mobility is neither too low nor too high. This highlights that policy effectiveness depends critically on the interaction between behavioral response and mobility, and cannot be fully understood when these mechanisms are considered in isolation.

This phenomenon does not arise in models that consider mobility or behavioral feedback in isolation. Instead, it is a direct consequence of their coupling, highlighting the importance of jointly modeling these mechanisms when evaluating epidemic control strategies.

C. Targeted interventions focused on the high-contact group

We evaluate targeted interventions aimed at the high-contact group (group 2), which plays a dominant role in transmission dynamics. Three strategies are considered: (i) increasing risk sensitivity (α_2), representing improved compliance or risk awareness; (ii) reducing mobility (m_2), representing targeted movement restrictions; and (iii) a combination of both.

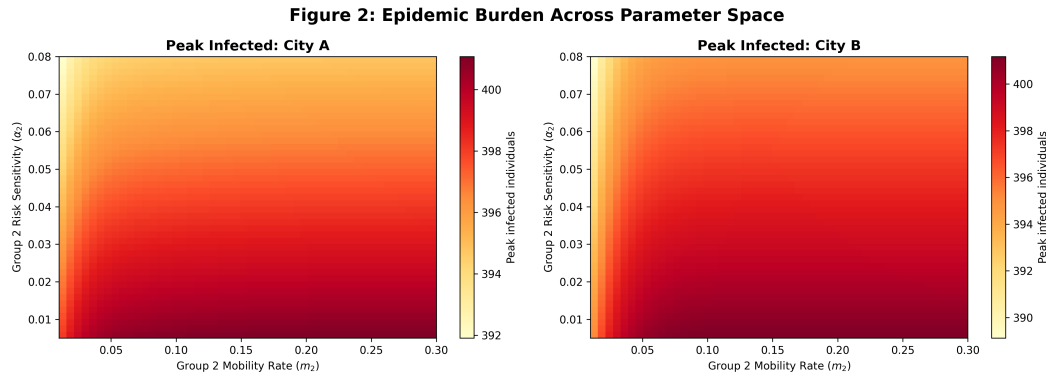


Fig. 2. Epidemic burden across parameter space. Heatmaps of peak infected individuals in (left) city A and (right) city B as functions of high-contact group mobility m_2 and risk sensitivity α_2 . Increasing α_2 reduces burden; increasing m_2 strengthens coupling and increases spillover.

Figure 3 shows that increasing α_2 reduces peak infection levels in both cities by accelerating behavioral adaptation. In contrast, reducing m_2 primarily delays and weakens cross-city transmission, with the strongest effect observed in the initially uninfected city.

The combined intervention produces the largest reduction in both peak burden and final epidemic size. This indicates a complementary effect: behavioral adaptation reduces transmission intensity, while mobility control limits spatial spread. Together, these mechanisms produce a stronger reduction than either intervention alone.

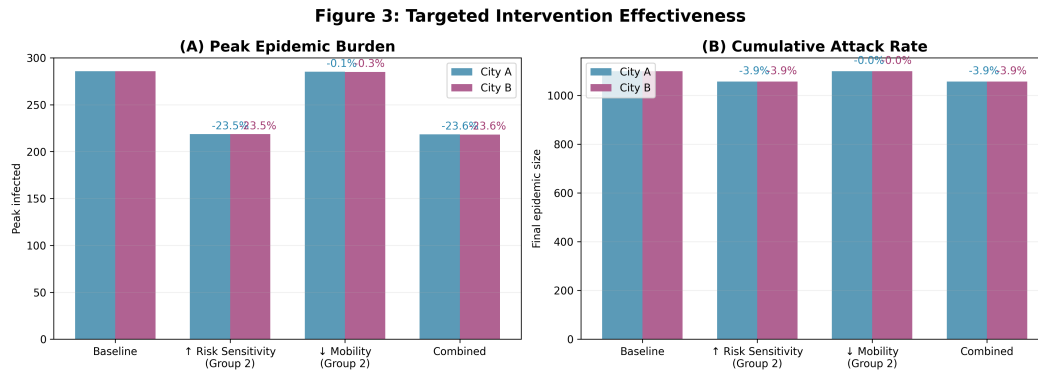


Fig. 3. Efficacy of targeted interventions. (A) Peak incidence and (B) total epidemic size under baseline conditions, increased risk sensitivity of group 2, reduced mobility of group 2, and the combination of both interventions. Targeting the high-contact group significantly reduces the epidemic, and combining interventions leads to the strongest reduction.

D. Supplementary: group dynamics and behavioral response

To gain some insight into how the system works from the inside, Supplementary Figure 4 illustrates the infection paths of each group and their contact rate dynamics. Note that the high-contact group drives early growth more, as it starts with a higher initial contact rate c_2^0 , while the cautious group has lower peaks. As the number of infections increases, people react and decrease their contacts, and this reaction is more significant if the group's behavioral sensitivity α_g is large.

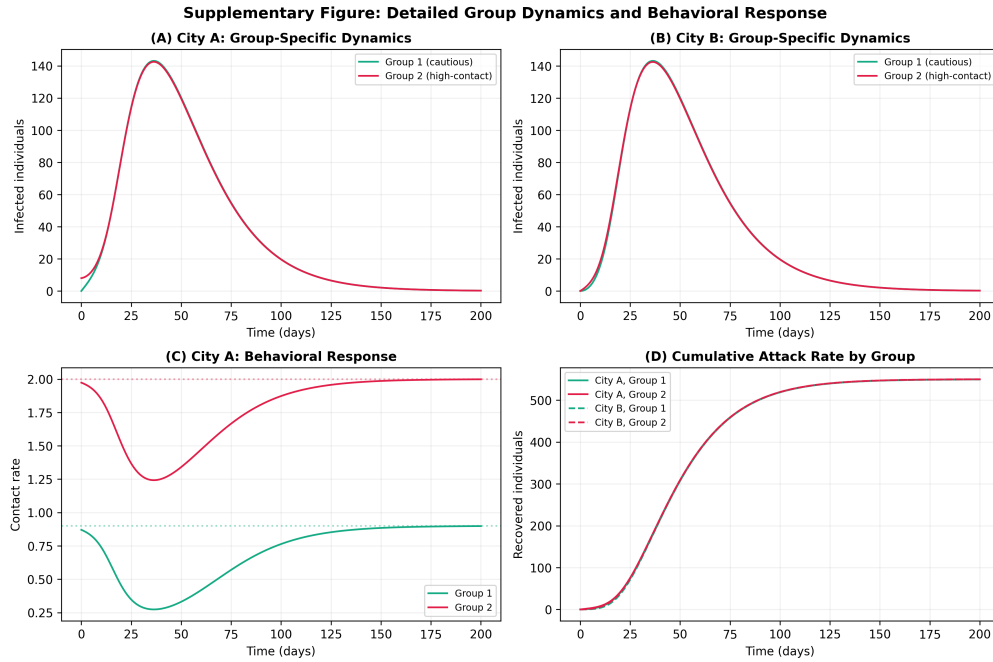


Fig. 4. Supplementary group-specific dynamics and behavioral response. This figure illustrates the group-level infection curves in each city, the response of contact rates over time with risk feedback, and the cumulative recovery totals over each group.

VI. DISCUSSION

What this study demonstrates is that the difference in human behavior and mobility patterns can significantly impact the course of an epidemic in ways that cannot be captured by a simple, deterministic model. There are three main points that can be gleaned from this study.

First, **mobility controls synchronization**. When there is little connectivity between two cities, one city will feel the effects of the epidemic before another city, effectively providing a window of preparation as the spillover is delayed. As connectivity increases, this spillover is hastened so that both cities feel the effects simultaneously, negating the preparation window.

Second, **behavioral feedback can substantially reduce peaks**, especially for that group with the most contact. The reason is that the infection pressure is determined by the weighted infected burden through $c_g^X(t)I_{Xg}(t)$, so that reducing contact in that group that initially has more contact intensity has significant effects. The heatmap suggests that there is a non-linear interaction between behavioral response and mobility, implying that small improvements in behavioral response can have large effects when mobility is intermediate.

Third, **targeted interventions are efficient**. Interventions that increase risk sensitivity, such as enhanced communication and support for compliance, or those that reduce mobility of the high-contact group, reduce both the peak and final size of the outbreak, with the combined approach being more effective. In this approach, mobility reduction for each group is understood broadly and does not necessarily mean universal travel bans.

A. Limitations and possible improvements

There are some limitations in the present study that suggest possible improvements in the future. The model we propose is deterministic, i.e., it cannot account for stochastic fadeouts, random introductions of new cases, or the timing of the peak [13]. Secondly, the model uses a prevalence-driven exponential response to reduce contacts. It may be worth exploring other forms of response, such as saturating, delayed, and incidence-driven response [10]. Mixing is homogeneous within cities, with no age

structure, occupation network, or explicit assortativity. Extensions could include cross-group contact matrices, asymmetric city sizes with directional mobility, endogenous policy triggers, and calibration to surveillance or mobility datasets.

In addition, communication dynamics are not modeled explicitly. Coupled awareness–disease processes on multilayer social/contact structures can shift effective epidemic thresholds and alter intervention leverage points [11]. Embedding those mechanisms would provide a richer representation of information-driven adaptation.

B. Publication and reproducibility statements

Data availability. This study is simulation based and does not use individual-level human-subject data. All model parameters, equations, and outcome metrics are documented in the manuscript and appendix.

Code availability. The full workflow required to reproduce figures and parameter sweeps consists of deterministic ODE integration and post-processing steps described in Section IV; scripts can be archived alongside this manuscript upon submission.

Ethics statement. No human participants, identifiable data, or animal experiments were involved.

Conflict of interest. The authors declare no competing interests.

VII. CONCLUSION AND FUTURE WORK

We proposed a two-city, two-group SIR framework that integrates heterogeneous risk-perception feedback and group-specific mobility in a single analyzable system. Using a next-generation matrix approach, we computed invasion thresholds and explored regime changes across mobility intensities. Results indicate that mobility primarily governs spillover speed and synchronization, while behavioral sensitivity—especially in the high-contact group—governs peak suppression and final size. Targeted interventions that increase risk sensitivity and/or reduce mobility in the high-contact group are consistently the most effective, and combined strategies provide the largest reductions. Overall, the framework provides a tractable bridge between threshold analysis and rapid scenario exploration in metapopulation epidemics.

VIII. ACKNOWLEDGMENT

I would like to express my sincere gratitude to my research supervisor, Shirali Khadyrov, for his valuable guidance, constructive feedback, and continuous support throughout the course of this study.

REFERENCES

- [1] R. M. Anderson and R. M. May, *Infectious Diseases of Humans: Dynamics and Control*. Oxford University Press, 1991.
- [2] J. Arino and P. van den Driessche, “A multi-city epidemic model,” *Mathematical Population Studies*, vol. 14, no. 3, pp. 185–203, 2007.
- [3] D. Balcan, V. Colizza, B. Gonçalves, H. Hu, J. J. Ramasco, and A. Vespignani, “Multiscale mobility networks and the spatial spreading of infectious diseases,” *Proceedings of the National Academy of Sciences*, vol. 106, no. 51, pp. 21484–21489, 2009.
- [4] C. T. Bauch and A. P. Galvani, “Social factors in epidemiology,” *Science*, vol. 342, no. 6154, pp. 47–49, 2013.
- [5] D. Brockmann and D. Helbing, “The hidden geometry of complex, network-driven contagion phenomena,” *Science*, vol. 342, no. 6164, pp. 1337–1342, 2013.
- [6] V. Colizza, A. Barrat, M. Barthélemy, and A. Vespignani, “The role of the airline transportation network in the prediction and predictability of global epidemics,” *Proceedings of the National Academy of Sciences*, vol. 103, no. 7, pp. 2015–2020, 2006.
- [7] O. Diekmann, J. A. P. Heesterbeek, and J. A. J. Metz, “On the definition and the computation of the basic reproduction ratio R_0 in models for infectious diseases in heterogeneous populations,” *Journal of Mathematical Biology*, vol. 28, pp. 365–382, 1990.
- [8] E. P. Fenichel *et al.*, “Adaptive human behavior in epidemiological models,” *Proceedings of the National Academy of Sciences*, vol. 108, no. 15, pp. 6306–6311, 2011.
- [9] S. Funk, E. Gilad, C. Watkins, and V. A. A. Jansen, “The spread of awareness and its impact on epidemic outbreaks,” *Proceedings of the National Academy of Sciences*, vol. 106, no. 16, pp. 6872–6877, 2009.
- [10] S. Funk, M. Salathé, and V. A. A. Jansen, “Modelling the influence of human behaviour on the spread of infectious diseases: A review,” *Journal of the Royal Society Interface*, vol. 7, no. 50, pp. 1247–1256, 2010.
- [11] C. Granell, S. Gomez, and A. Arenas, “Dynamical interplay between awareness and epidemic spreading in multiplex networks,” *Physical Review Letters*, vol. 111, no. 12, Art. no. 128701, 2013.

- [12] B. T. Grenfell and J. Harwood, "(meta)population dynamics of infectious diseases," *Trends in Ecology & Evolution*, vol. 12, no. 10, pp. 395–399, 1997.
- [13] M. J. Keeling and P. Rohani, *Modeling Infectious Diseases in Humans and Animals*. Princeton University Press, 2008.
- [14] W. O. Kermack and A. G. McKendrick, "A contribution to the mathematical theory of epidemics," *Proceedings of the Royal Society A*, vol. 115, no. 772, pp. 700–721, 1927.
- [15] N. Perra, D. Balcan, B. Goncalves, and A. Vespignani, "Towards a characterization of behavior-disease models," *PLoS ONE*, vol. 6, no. 8, Art. no. e23084, 2011.
- [16] P. Poletti, M. Ajelli, and S. Merler, "Risk perception and effectiveness of uncoordinated behavioral responses in an emerging epidemic," *Mathematical Biosciences*, vol. 238, no. 2, pp. 80–89, 2012.
- [17] P. van den Driessche and J. Watmough, "Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission," *Mathematical Biosciences*, vol. 180, pp. 29–48, 2002.
- [18] F. Verelst, L. Willem, and P. Beutels, "Behavioural change models for infectious disease transmission: A systematic review (2010–2015)," *Journal of the Royal Society Interface*, vol. 13, no. 125, Art. no. 20160820, 2016.
- [19] W. Wang and X.-Q. Zhao, "Threshold dynamics for compartmental epidemic models in periodic environments," *Journal of Dynamics and Differential Equations*, vol. 20, pp. 699–717, 2008.