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Reconstructed physical network for disease spread using age-based contact data: Simulations of case studies across Chinese regions

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Abstract

In reality, contact patterns among individuals vary by age. To examine disease transmission across different age groups, this study builds a transmission network using authentic population proportions and age-specific matrices of contact frequencies. In addition, to describe the transmission effect of different network structures, this paper uses an edge reconnecting algorithm to construct a random network and a Barabási-Albert (BA) network, both matching the age-specific contact frequency matrices from the real dataset. Based on those networks, we develop the two-layer coupled $UA_nA_pU - SEIR$ model, and then conduct threshold analysis for this epidemic model using the microscopic Markov chain approach (MMCA). To validate our analytical findings, we further compare the numerical outputs from our simulations against real-world provincial-level infection data across China. The results indicate that the reconstructed disease transmission networks can mathematically reflect outbreak thresholds. Meanwhile, the two networks reconstructed from random and BA baseline networks can, in the mathematical derivation of outbreak thresholds, respectively serve as network models for regions with lower and higher levels of economic development. Moreover, given equal disease impact on all individuals, younger people face a higher infection risk due to their distinct behavioral contact patterns compared to other groups. Hence, this study can provide practical guidance for practitioners in building more realistic disease transmission networks and managing epidemic spread.

Keywords: Multiplex network, Age-stratified contact matrices, Epidemic transmission, Model comparison, Compartmental epidemic model

1 Introduction

Since Kermack and McKendrick introduced the SIR model to describe disease transmission in 1927[1], scholars have widely adopted mathematical models to derive outbreak thresholds and investigate epidemic dynamics in real-world settings. Later, to address major outbreaks like SARS and COVID-19, researchers extended the SIR model into SIS, SEIR, and SVIR variants to better reflect different disease dynamics[2–4]. Subsequently, the use of small-world[5] and scale-free networks[6] laid a foundation for exploring how diseases spread within communities. Hence, mathematical models of infectious diseases have gradually aligned more closely with real-world conditions, with individual heterogeneity emerging as a core focus of this refinement process.

During the study of disease spread, individual heterogeneity is considered an important parameter when constructing epidemic transmission models. Methods that do not account for such heterogeneity are referred to as homogeneous mixing[7]. Under homogeneous mixing, interactions between any two individuals are assumed to occur with equal probability, which means they are without heterogeneity. In the study of HIV transmission, May et al.[8] recognized that highly active individuals in a community can spread the disease more effectively than others. This finding stands as a key early marker of heterogeneity being incorporated into the study of the epidemic transmission process. Subsequently, such heterogeneity-based methods were also extended to the research of vector-borne disease transmission[9].

Beyond individual-level transmission heterogeneity, researchers have further identified that the local environment where individuals reside also shapes propagation dynamics, with environmental pressure being one of the most well-documented examples. Festinger et al.[10] found that an individual’s judgments depend on their own insights and the behaviors of those nearby. This influence, which comes from neighbors in the same environment, is called environmental pressure. In subsequent studies, environmental pressure as an individual heterogeneity played an important role in transmission processes such as vaccination[11] and rumor spreading[12].

Building on these studies of individual and environmental heterogeneity, researchers have further sought to embed heterogeneous characteristics directly into the topological structure of contact networks. The most intuitive reflection of network heterogeneity is to specify which individuals in the network can form complete connections with each other[13, 14]. For instance, Zhou et al.[15] construct a heterogeneous network by representing recurrent events as multi-dimensional Hawkes processes. To accommodate the development of communication, activity-driven networks[16] have been employed as network structures for the disease transmission process. Yue et al.[17] modified the disease transmission layer using an activity-driven network, allowing its structure to evolve over time. To better represent heterogeneity in real-world networks,

some scholars have introduced the concept of simplicial complexes into activity-driven networks[18, 19].

Among all the factors that shape contact heterogeneity, age has long been recognized as one of the most critical, as it directly determines how individuals interact with each other. Initially, age was incorporated as a heterogeneity factor into models of disease transmission to study childhood diseases[20]. However, most prior studies simply treat age-specific contact patterns as a single probability within disease transmission. These studies have simply treated age-specific contact patterns as a single model parameter, without conducting more research into this dimension. In fact, the way contacts vary among age groups should be modeled as a full network in transmission studies, not simply inserted into a probability equation. Incorporating contact patterns into a preconstructed transmission network for disease spread studies would disrupt both the original network’s propagation dynamics and the age-specific contact patterns. Therefore, it is necessary to consider how to reconstruct the age-contact patterns as a complete network.

Consequently, we consider constructing a heterogeneous network based on the population’s age structure and the matrix of contact frequencies between age groups, which we will use to investigate the spread of diseases. Here, we use an existing dataset[21] to construct random networks and BA networks that follow the population’s age structure and the age contact frequency matrix reported in the data[21]. We then compare these networks with the ordinary BA networks that are commonly used in epidemic transmission spreading models. To accurately compare the differences among network structures, we employ MMCA to construct a two-layer network and analyze the outbreak threshold[22]. We introduce a two-layer network that represents information spreading and disease transmission, respectively. To better capture real-world disease spread with an incubation period, such as SARS and H1N1[23–25], we characterize the process using the SEIR model. Unlike disease transmission, an individual’s information uptake is shaped by social pressures from neighbors in their community[10]. As a result, to capture the effect of environmental pressure on information propagation, we use the standard Heaviside step function to model how this pressure shapes the propagation dynamics within the information transmission layer. Based on the above analysis, we propose the $UA_nA_pU - SEIR$ coupled model for mathematical simulations. Using the MMCA approach, we analyze how this model evolves under different network structures.

The remainder of this paper is organized as follows. Section 2 introduces the age-dataset, the associated network modeling, and the mathematical formulation used to simulate epidemic spread. Section 3 applies MMCA to analyze the disease transmission model. Section 4 performs extensive numerical simulations to compare the dynamic behavior of the propagation model across various network structures. Section 5 concludes with a summary and an outlook on future research.

2 Model description

2.1 Modeling population contact patterns

Depending on the age-specific contact frequency matrix and different diseases, network structures can be built in several ways. In this work, we assume that the disease does not noticeably alter individual behavior and that it leads to the same outcome for all infected people. Consequently, we can focus on how the disease spreads dynamically under different network models.

In this paper, we employ an edge reconnecting model[26] to reconstruct the corresponding age contact network. Compared with other network reconfiguration methods, the edge reconnecting method has the property of strictly preserving the degree of each individual node. This ensures that our network reconstruction process will not alter the original degree distribution of the baseline network. For instance, the edge reconnecting method can perfectly retain the original distribution of hub nodes in BA networks. Hence, this method enables us to investigate disease transmission dynamics under different network topologies in our subsequent research. Before performing the reconnecting, we first prepare an existing network, and assign an age to each node according to the age distribution of the population reported in dataset[21]. At each time step t , we randomly select two edge pairs in the network, such as $E(v_1, w_1)$ and $E(v_2, w_2)$. We then swap their endpoints, changing the connections to $E(v_1, w_2)$ and $E(v_2, w_1)$ without altering the degree of any node, as shown in Figure 1. Next, we compare the generated network at step t with the one at step $t - 1$. If the age contact frequency matrix derived from the network at step t is closer to the target matrix provided in the dataset[21] than that from step $t - 1$, we keep the network at step t . Otherwise, we retain the network from step $t - 1$. This process continues until the maximum percentage difference error between the age contact frequency matrix and the target matrix drops below 5%.

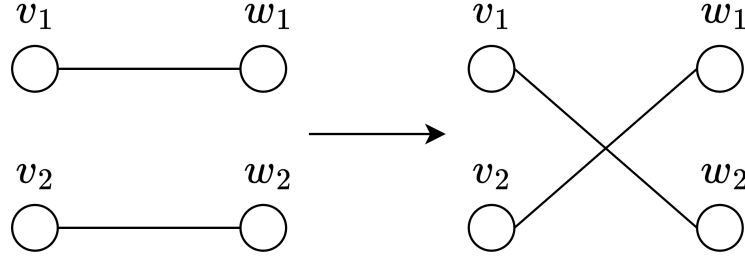


Fig. 1 Edge reconnecting model. A basic illustration of the edge reconnecting algorithm, where modifications are reversed if the result deviates from expectations.

To enable subsequent network modeling, we draw on the data reported by Dina et al.[21]. That dataset stores various population contact patterns and related information for Australia, Canada, China, India, Israel, Japan, Russia, South Africa, and the United States of America. The dataset accurately records, for each country and

region, the age-specific population proportions and the contact frequencies between different age groups. Moreover, it stratifies these contacts into five settings: household, community, workplace, school, and overall. In the subsequent analysis, we use the national age contact frequency matrix and the age-specific population proportion data for China to construct our model, as shown in Figure 2.

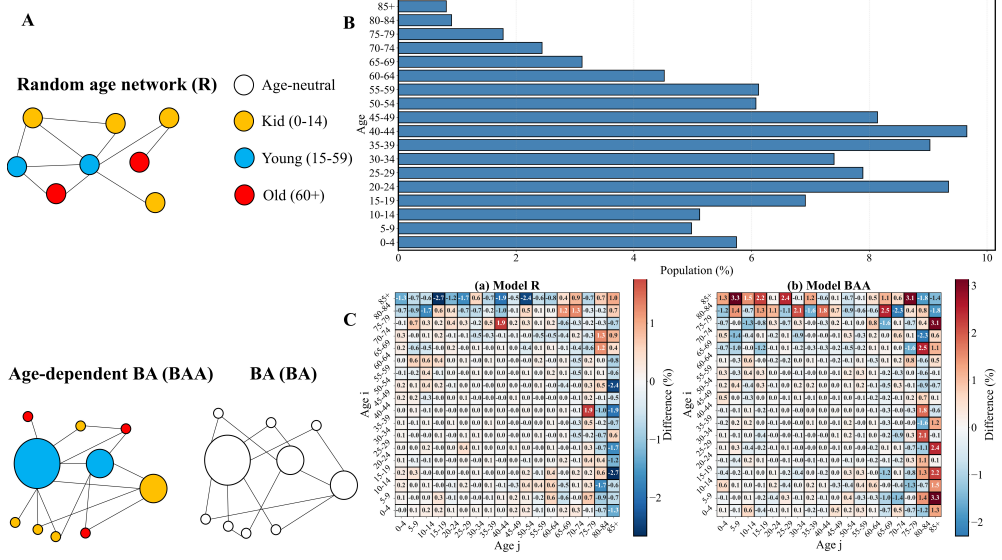


Fig. 2 Modeling age contact networks. Panel A: The figure shows the different constructed models. Using edge reconnecting method, we reconstruct both a random network and a BA network based on the age contact frequency matrix and the associated age distribution data from the dataset[21]. The reconstructed random network is denoted as Model R, and the reconstructed BA network as Model BAA. In addition, because BA networks are widely used in disease transmission studies, we also prepared an unmodified BA network as Model BA for comparison with other research. Panel B: Population proportions across age groups in China, as reported by Dina et al.[21]. Panel C: Comparison of Model R and Model BAA with the age contact frequency matrix from Dina et al.[21].

Figure 2B presents the demographic proportions of China's national population used in this research[21]. Figure 2A shows the network structures generated from the population age dataset and contact frequency matrix. Based on the above discussion, we first assign an age to each node of a random network, placing every node into an age group. Then we apply the edge reconnecting algorithm described earlier to this random network. The resulting network, whose age contact frequency matrix meets expectations, is referred to as Model R. Similarly, we apply the same procedure to a BA network, and the resulting network is designated as Model BAA. For comparison with Model R and Model BAA, we also include a BA network of exactly the same size, referred to as Model BA. This model is not subjected to the edge reconnecting method and serves only as a baseline for comparative experiments. To verify the accuracy of the constructed models, Figure 2C compares the age contact frequency matrices derived from Model R and Model BAA with the target matrix given by the

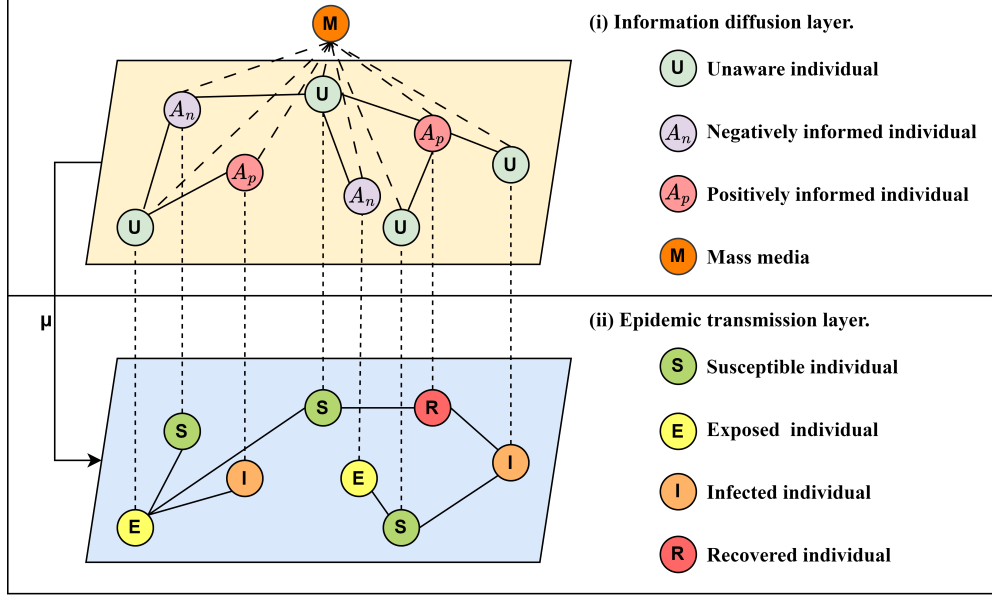


Fig. 3 The framework of the $UA_n A_p U - SEIR$ model. Information diffusion will be described in the first layer. The second layer depicts disease spread. Between layers, dotted lines are used to map node relationships across distinct networks. Furthermore, within the first layer, extra dotted lines represent the impact of mass media on individual nodes.

dataset[21]. Using the relative percentage difference method, the matrices of Model R and Model BAA are compared with the target matrix from the dataset. In both cases, the maximum error is below 4%. Additionally, the edge reconnecting method preserves the degree of each individual node during the reconfiguration process. For Model R and Model BAA, this means the degrees of their nodes remain consistent with those of their respective baseline networks: a random network and a BA network. Hence, this maximally preserves the original structural characteristics of the baseline networks.

2.2 Modeling Infectious Disease Spread

To further explore the transmission dynamics of these three models in disease spread, we first consider constructing a two-layer coupled model using MMCA. In real life, the spread of epidemics often comes with both official information and rumors. Hence, we propose a two-layer coupled model $UA_n A_p U - SEIR$, which incorporates information diffusion and disease transmission. Figure 3 presents the structure of the two-layer model. The virtual network component captures the dissemination of formal official alerts and unsubstantiated rumors in the first layer, while the physical contact network describes the diffusion of the epidemic among individuals in the second layer. Moreover, the contact network derived from 2.1 governs the disease spread in layer two.

For the first network layer, all individuals fall into three states: unaware (U), rumor-accepting (A_n), and those who trust the official guidance (A_p). We posit that unaware individuals can update their state after receiving information from neighbors.

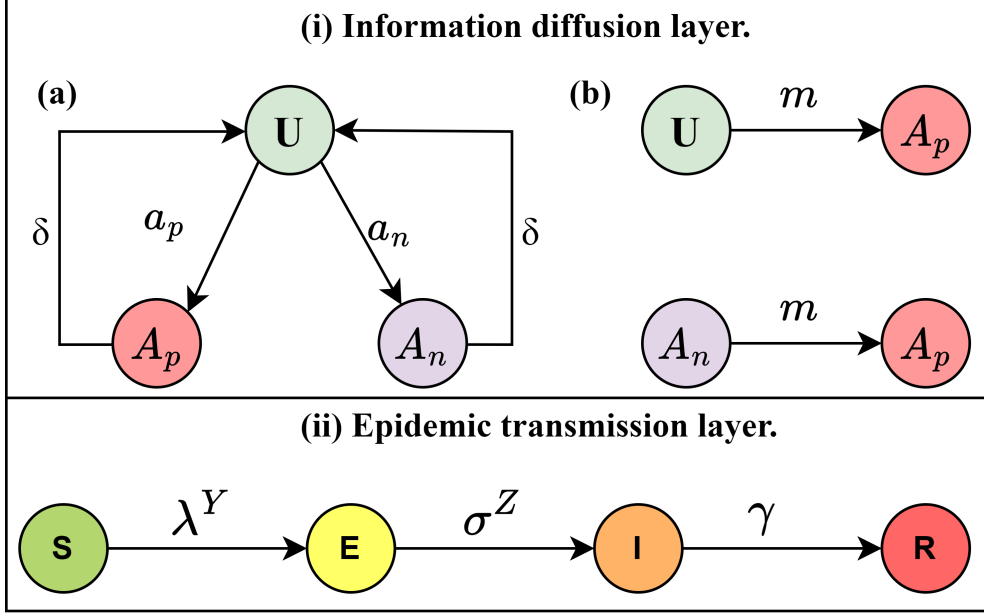


Fig. 4 Transition rules within the information diffusion layer and the epidemic transmission layer. Panel (i) illustrates how official guidance and rumors propagate over time. Panel (ii) depicts the diffusion process of disease.

Specifically, interacting with an A_p neighbor triggers a switch to A_p with probability α_p , while contact with an A_n neighbor drives a transition to A_n with probability α_n . Additionally, individuals in A_p or A_n may forget the content and revert to U with probability δ . We also incorporate mass media M , which broadcasts official messages to all individuals with probability m , converting any U or A_n individuals directly to A_p . This spreading dynamics is visualized in Figure 4.

The process of individuals receiving information differs from that of disease infection. Specifically, an individual can only be exposed to information when its local prevalence in the surrounding environment reaches a specific threshold[27, 28]. This process exhibits distinct step-like behavior, meaning the Heaviside step function is typically used to mathematically capture the effect of environmental pressure on individuals. To reflect that environmental pressure affects information uptake, we modify the transmission probabilities α_p and α_n . Specifically, we introduce Ω_i^p and Ω_i^n to represent the pressure exerted by neighbors on individual i . We define $F(X) = 1$ when $X > 0$, and $F(X) = 0$ otherwise. The dynamic pressure factors Ω_i^p and Ω_i^n are then given by:

$$\begin{cases} \Omega_i^p = F\left\{\frac{\sum_j^N a_{ij}[P_j^{A_p}(t) - P_j^{A_n}(t)]}{k_i^a}\right\} \\ \Omega_i^n = F\left\{\frac{\sum_j^N a_{ij}[P_j^{A_n}(t) - P_j^{A_p}(t)]}{k_i^a}\right\} \end{cases} \quad (1)$$

where a_{ij} denote the elements of the adjacency matrix in the information propagation layer, and let k_i^a represent the number of neighbors of node i in that layer. $P_j^{A_p}(t)$

and $P_j^{A_n}(t)$ are, respectively, the probabilities that individual j is in the A_p or A_n state at time t . At each time step t , individual i can switch to state A_p or A_n with the basic probability of information transmission α_0 only if, among all neighbors in its environment, the probability of one state (either A_p or A_n) exceeds that of the other. Consequently, the transition probabilities α_p and α_n are expressed as follows:

$$\begin{cases} \alpha_p = \alpha_0 \Omega_i^p \\ \alpha_n = \alpha_0 \Omega_i^n \end{cases} \quad (2)$$

The second layer is dedicated to modeling epidemic transmission, which follows the SEIR framework. Under this process, each individual can fall into one of four states: susceptible (S), who have not yet contracted the disease, exposed (E), who have been infected but are not yet infectious, infectious (I), who are able to transmit the disease to others, and recovered (R), who have cleared the infection. When a susceptible individual (S) comes into contact with an infected neighbor, it moves to the exposed state (E) with probability λ^Y . Furthermore, an exposed individual (E) progresses to the infected state (I) with probability σ^Z . Finally, an individual in state I will transition to the recovered state (R) with probability γ . This process is illustrated in [Figure 4](#).

In real life, there is a dynamic correlation between information spread and disease transmission. For instance, individuals who believe rumors may think they are protecting themselves properly, thus overlooking the real threat of the disease. By contrast, those who trust official information tend to adopt correct behaviors, such as wearing masks, which lowers their infection risk. To reflect this effect, we introduce a decay factor μ with $\mu \in [0, 1]$. Within the epidemic transmission layer, μ describes how different information states lead to distinct probabilities of infection:

$$\lambda^Y = \begin{cases} \lambda_0 & , Y = A_n S \\ \mu \lambda_0 & , Y = U S \\ \mu^2 \lambda_0 & , Y = A_p S \end{cases} \quad (3)$$

where λ_0 stands for the baseline transmission rate. Meanwhile, the attenuation factor μ serves an equivalent function in the state transition probability between the exposed and infectious statuses. As a result, the transition rate σ^Z can be formulated as follows:

$$\sigma^Z = \begin{cases} \sigma_0 & , Z = A_n E \\ \mu \sigma_0 & , Z = U E \\ \mu^2 \sigma_0 & , Z = A_p E \end{cases} \quad (4)$$

where σ_0 stands for the baseline transition rate. All the relevant parameter notations are detailed in [Table 1](#).

Table 1 Parameter definitions.

Parameter	Definition
a_{ij}	Adjacency matrix of the information diffusion layer.
b_{ij}	Adjacency matrix for the disease transmission layer.
α_p	Rate of transition from U to A_p after receiving official information.
α_n	Rate of transition from U to A_n after receiving rumors.
δ	The forgetting rate of rumors or official information, which leads A_n or A_p to return to U .
m	Mass media's rate of spreading official information.
λ_0	Basic infection rate from state S to state E .
σ_0	Basic transition rate from state E to state I .
μ	The attenuation factor of disease infection or exposure.
γ	The epidemic recovery rate for state I .

3 Theoretical investigation based on the MMCA

To further compare different age-related networks, we employ the MMCA to mathematically study the $UA_nA_pU-SEIR$ two-layer coupled model and derive its outbreak threshold. To simplify the two-layer coupled model and ensure the spread process is more realistic, we assume that individuals transition to the state I in the disease transmission layer, they actively seek official information in hopes of recovering faster. Hence, they will change to the A_p state in the information diffusion layer. Meanwhile, we consider that recovered individuals (R) have fully grasped the correct protective measures during their illness. Although they may forget some protective details, these straightforward behaviors, including mask-wearing in hazardous places such as hospitals, remain intact. Hence, they will not change to state A_n in the first layer. Thus, at time step t , each node in this model can be in nine states: $US, UE, UR, A_nS, A_nE, A_pS, A_pE, A_pI$, and A_pR . The valid node states are provided in Table 1. The probabilities that individual i occupies these nine states at time step t are denoted as $P_i^{US}(t), P_i^{UE}(t), P_i^{UR}(t), P_i^{A_nS}(t), P_i^{A_nE}(t), P_i^{A_pS}(t), P_i^{A_pE}(t), P_i^{A_pI}(t)$, and $P_i^{A_pR}(t)$, respectively, satisfying the normalization condition $P_i^{US}(t) + P_i^{UE}(t) + P_i^{UR}(t) + P_i^{A_nS}(t) + P_i^{A_nE}(t) + P_i^{A_pS}(t) + P_i^{A_pE}(t) + P_i^{A_pI}(t) + P_i^{A_pR}(t) = 1$.

Let a_{ij} and b_{ij} represent the adjacency matrix entries of the information diffusion layer and the disease transmission layer, respectively. At each time step t , we denote $r_i^{A_n}(t)$ and $r_i^{A_p}(t)$ as the probabilities that an individual in state U within the information diffusion layer is not influenced by neighbors in state A_n or A_p into changing to the corresponding state. Meanwhile, for the epidemic transmission layer, we let $q_i^Y(t)$ be the probability that individual i in state S is not infected by any neighbor in state I and thus does not transition to state E , where $Y \in \{US, A_nS, A_pS\}$. According to MMCA, we obtain:

$$r_i^{A_n}(t) = \prod_j [1 - a_{ji} P_j^{A_n}(t) \alpha_n] \quad (5)$$

Table 2 Individual in each state of the $UA_nA_pU - SEIR$ model

US	Susceptible and unaware of disease-related information.	UE	In the exposed state and unaware of disease-related information.	UR	Recovered and unaware of disease-related information.
A_nS	Susceptible with a negative attitude toward official epidemic information.	A_nE	In the exposed state with a negative attitude toward official epidemic information.	A_pS	Susceptible with a positive attitude toward official epidemic information.
A_pE	In the exposed state with a positive attitude toward official epidemic information.	A_pI	Infected with a positive attitude toward official epidemic information.	A_pR	Recovered with a positive attitude toward official epidemic information.

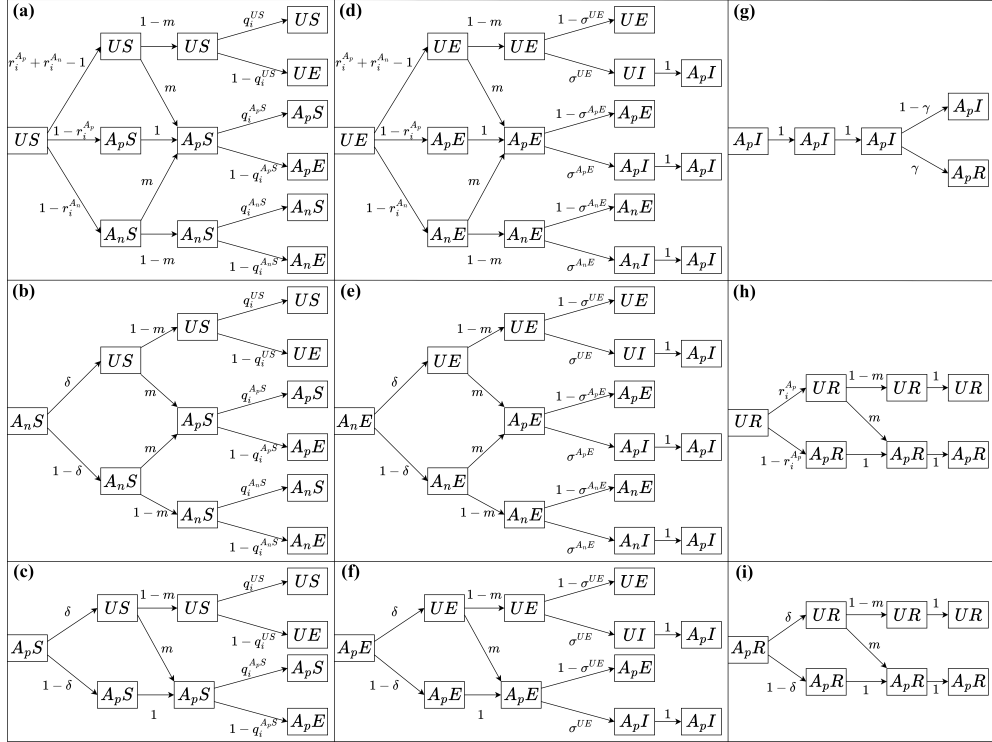


Fig. 5 Tree diagrams of state transition probabilities for each effective status. (a)- US , (b)- A_nS , (c)- A_pS , (d)- UE , (e)- A_nE , (f)- A_pE , (g)- A_pI , (h)- UR , (i)- A_pR

$$r_i^{A_p}(t) = \prod_j [1 - a_{ji} P_j^{A_p}(t) \alpha_p] \quad (6)$$

$$q_i^Y(t) = \prod_j [1 - b_{ji} P_j^I(t) \lambda^Y], \quad Y \in \{US, A_nS, A_pS\} \quad (7)$$

Drawing on the analysis above, we can construct the state transition probability tree under the MMCA, as visualized in [Figure 5](#). Using this tree structure, we derive the state transition equations for every individual status via the MMCA approach:

$$\begin{aligned} P_i^{A_pE}(t+1) = & P_i^{US}(t)[1 - q_i^{A_pS}(t)]m[r_i^{A_p}(t) + r_i^{A_n} - 1] + P_i^{US}(t)[1 - q_i^{A_pS}(t)][1 - r_i^{A_p}(t)] \\ & + P_i^{US}(t)[1 - q_i^{A_pS}(t)]m[1 - r_i^{A_p}(t)] + P_i^{UE}(t)(1 - \sigma^{A_pE})m[r_i^{A_p}(t) + r_i^{A_n}(t) - 1] \\ & + P_i^{UE}(t)(1 - \sigma^{A_pE})[1 - r_i^{A_p}(t)] + P_i^{UE}(t)(1 - \sigma^{A_pE})m[1 - r_i^{A_n}(t)] \\ & + P_i^{A_nS}(t)[1 - q_i^{A_pS}(t)]m\delta + P_i^{A_nS}(t)[1 - q_i^{A_pS}(t)]m(1 - \delta) \\ & + P_i^{A_nE}(t)(1 - \sigma^{A_pE})m\delta + P_i^{A_nE}(t)(1 - \sigma^{A_pE})m(1 - \delta) \\ & + P_i^{A_pS}(t)[1 - q_i^{A_pS}(t)]m\delta + P_i^{A_pS}(t)[1 - q_i^{A_pS}(t)](1 - \delta) \\ & + P_i^{A_pE}(t)(1 - \sigma^{A_pE})m\delta + P_i^{A_pE}(t)(1 - \sigma^{A_pE})(1 - \delta) \end{aligned} \quad (8)$$

$$\begin{aligned} P_i^{US}(t+1) = & P_i^{US}(t)q_i^{US}(t)(1 - m)[r_i^{A_p}(t) + r_i^{A_n}(t) - 1] + P_i^{A_nS}(t)q_i^{US}(t)(1 - m)\delta \\ & + P_i^{A_pS}(t)q_i^{US}(t)(1 - m)\delta \end{aligned} \quad (9)$$

$$\begin{aligned} P_i^{UE}(t+1) = & P_i^{UE}(t)(1 - \sigma^{UE})(1 - m)[r_i^{A_n}(t) + r_i^{A_p}(t) - 1] \\ & + P_i^{US}(t)[1 - q_i^{US}(t)](1 - m)[r_i^{A_p}(t) + r_i^{A_n}(t) - 1] \\ & + P_i^{A_nS}(t)[1 - q_i^{US}(t)](1 - m)\delta + P_i^{A_nE}(t)(1 - \sigma^{UE})(1 - m)\delta \\ & + P_i^{A_pS}(t)[1 - q_i^{US}(t)](1 - m)\delta + P_i^{A_pE}(t)(1 - \sigma^{UE})(1 - m)\delta \end{aligned} \quad (10)$$

$$P_i^{UR}(t+1) = P_i^{UR}(t)(1 - m)r_i^{A_p}(t) + P_i^{UR}(t)(1 - m)\delta \quad (11)$$

$$P_i^{A_nS}(t+1) = P_i^{US}(t)q_i^{A_nS}(t)(1 - m)[1 - r_i^{A_n}(t)] + P_i^{A_nS}(t)q_i^{A_nS}(t)(1 - m)(1 - \delta) \quad (12)$$

$$\begin{aligned} P_i^{A_nE}(t+1) = & P_i^{US}(t)[1 - q_i^{A_nS}(t)](1 - m)[1 - r_i^{A_n}(t)] \\ & + P_i^{UE}(t)(1 - \sigma^{A_nE})(1 - m)[1 - r_i^{A_n}(t)] \\ & + P_i^{A_nS}(t)[1 - q_i^{A_nS}(t)](1 - m)(1 - \delta) \\ & + P_i^{A_nE}(t)(1 - \sigma^{A_nE})(1 - m)(1 - \delta) \end{aligned} \quad (13)$$

$$\begin{aligned} P_i^{A_pR}(t+1) = & P_i^{UR}(t)mr_i^{A_p}(t) + P_i^{UR}(t)[1 - r_i^{A_p}(t)] + P_i^{A_pR}(t)m\delta + P_i^{A_pR}(t)(1 - \delta) \\ & + P_i^{A_pI}(t)\gamma \end{aligned} \quad (14)$$

$$\begin{aligned}
P_i^{A_p S}(t+1) = & P_i^{US}(t)q_i^{A_p S}(t)m[r_i^{A_p}(t) + r_i^{A_n}(t) - 1] + P_i^{US}(t)q_i^{A_p S}(t)[1 - r_i^{A_p}(t)] \\
& + P_i^{US}(t)q_i^{A_p S}(t)m[1 - r_i^{A_n}(t)] + P_i^{A_n S}(t)q_i^{A_p S}(t)m\delta \\
& + P_i^{A_n S}(t)q_i^{A_p S}(t)m(1 - \delta) + P_i^{A_p S}(t)q_i^{A_p S}(t)m\delta \\
& + P_i^{A_p S}(t)q_i^{A_p S}(t)(1 - \delta)
\end{aligned} \tag{15}$$

$$\begin{aligned}
P_i^{A_p I}(t+1) = & P_i^{UE}(t)\sigma^{UE}(1 - m)[r_i^{A_p}(t) + r_i^{A_n}(t) - 1] \\
& + P_i^{UE}(t)\sigma^{A_p E}m[r_i^{A_p}(t) + r_i^{A_n}(t) - 1] \\
& + P_i^{UE}(t)\sigma^{A_p E}[1 - r_i^{A_p}(t)] + P_i^{UE}(t)\sigma^{A_p E}m[1 - r_i^{A_n}(t)] \\
& + P_i^{UE}(t)\sigma^{A_n E}(1 - m)[1 - r_i^{A_n}(t)] + P_i^{A_n E}(t)\sigma^{UE}(1 - m)\delta \\
& + P_i^{A_n E}(t)\sigma^{A_p E}m\delta + P_i^{A_n E}(t)\sigma^{A_p E}m(1 - \delta) \\
& + P_i^{A_n E}(t)\sigma^{A_n E}(1 - m)(1 - \delta) + P_i^{A_p E}(t)\sigma^{UE}(1 - m)\delta \\
& + P_i^{A_p E}(t)\sigma^{A_p E}m\delta + P_i^{A_p E}(t)\sigma^{A_p E}(1 - \delta) + p_i^{A_p I}(t)(1 - \gamma)
\end{aligned} \tag{16}$$

At steady state, the equalities $P_i(t+1) = P_i(t) = P_i$ hold for all states of each individual i . Meanwhile, the probability that individual i becomes infected approaches 0. Hence we can set $P_i^I = P_i^{A_p I} = \varepsilon_i < 1$. Consequently, (7) can be approximated as:

$$q_i^Y = 1 - \lambda^Y \sum_j (b_{ji}\varepsilon_j), Y \in \{US, A_n S, A_p S\} \tag{17}$$

Substituting (17) into (8) - (16) yields:

$$\begin{aligned}
P_i^{A_p E} = & P_i^{US}m(r_i^{A_p} + r_i^{A_n} - 1)\lambda^{A_p S} \sum_j (b_{ji}\varepsilon_j) + P_i^{US}(1 - r_i^{A_p})\lambda^{A_p S} \sum_j (b_{ji}\varepsilon_j) \\
& + P_i^{US}m(1 - r_i^{A_n})\lambda^{A_p S} \sum_j (b_{ji}\varepsilon_j) + P_i^{UE}(1 - \sigma^{A_p E})m(r_i^{A_p} + r_i^{A_n} - 1) \\
& + P_i^{UE}(1 - \sigma^{A_p E})(1 - r_i^{A_p}) + P_i^{UE}(1 - \sigma^{A_p E})m(1 - r_i^{A_n}) \\
& + P_i^{A_n S}m\delta\lambda^{A_p S} \sum_j (b_{ji}\varepsilon_j) + P_i^{A_n S}m(1 - \delta)\lambda^{A_p S} \sum_j (b_{ji}\varepsilon_j) \\
& + P_i^{A_n E}(1 - \sigma^{A_p E})m\delta + P_i^{A_n E}(1 - \sigma^{A_p E})m(1 - \delta) \\
& + P_i^{A_p S}m\delta\lambda^{A_p S} \sum_j (b_{ji}\varepsilon_j) + P_i^{A_p S}(1 - \delta)\lambda^{A_p S} \sum_j (b_{ji}\varepsilon_j) \\
& + P_i^{A_p E}(1 - \sigma^{A_p E})m\delta + P_i^{A_p E}(1 - \sigma^{A_p E})(1 - \delta)
\end{aligned} \tag{18}$$

$$P_i^{US} = P_i^{US}(1 - m)(r_i^{A_p} + r_i^{A_n} - 1) + P_i^{A_n S}(1 - m)\delta + P_i^{A_p S}(1 - m)\delta \tag{19}$$

$$\begin{aligned}
P_i^{UE} = & P_i^{UE}(1 - \sigma^{UE})(1 - m)(r_i^{A_n} + r_i^{A_p} - 1) \\
& + P_i^{US}(1 - m)(r_i^{A_p} + r_i^{A_n} - 1)\lambda^{US} \sum_j (b_{ji}\varepsilon_j) \\
& + P_i^{A_nS}(1 - m)\delta\lambda^{US} \sum_j (b_{ji}\varepsilon_j) + P_i^{A_nE}(1 - \sigma^{UE})(1 - m)\delta \\
& + P_i^{A_pS}(1 - m)\delta\lambda^{US} \sum_j (b_{ji}\varepsilon_j) + P_i^{A_pE}(1 - \sigma^{UE})(1 - m)\delta
\end{aligned} \tag{20}$$

$$P_i^{UR} = P_i^{UR}(1 - m)r_i^{A_p} + P_i^{UR}(1 - m)\delta \tag{21}$$

$$P_i^{A_nS} = P_i^{US}(1 - m)(1 - r_i^{A_n}) + P_i^{A_nS}(1 - m)(1 - \delta) \tag{22}$$

$$\begin{aligned}
P_i^{A_nE} = & P_i^{US}(1 - m)(1 - r_i^{A_n})\lambda^{A_nS} \sum_j (b_{ji}\varepsilon_j) + P_i^{UE}(1 - \sigma^{A_nE})(1 - m)(1 - r_i^{A_n}) \\
& + P_i^{A_nS}(1 - m)(1 - \delta)\lambda^{A_nS} \sum_j (b_{ji}\varepsilon_j) + P_i^{A_nE}(1 - \sigma^{A_nE})(1 - m)(1 - \delta)
\end{aligned} \tag{23}$$

$$P_i^{A_pR} = P_i^{UR}mr_i^{A_p} + P_i^{UR}(1 - r_i^{A_p}) + P_i^{A_pR}m\delta + P_i^{A_pR}(1 - \delta) \tag{24}$$

$$\begin{aligned}
P_i^{A_pS} = & P_i^{US}m(r_i^{A_p} + r_i^{A_n} - 1) + P_i^{US}(1 - r_i^{A_p}) + P_i^{US}m(1 - r_i^{A_n}) \\
& + P_i^{A_nS}m\delta + P_i^{A_nS}m(1 - \delta) + P_i^{A_pS}m\delta + P_i^{A_pS}(1 - \delta)
\end{aligned} \tag{25}$$

$$\begin{aligned}
\varepsilon_i = & P_i^{UE}\sigma^{UE}(1 - m)(r_i^{A_p} + r_i^{A_n} - 1) + P_i^{UE}\sigma^{A_pE}m(r_i^{A_p} + r_i^{A_n} - 1) \\
& + P_i^{UE}\sigma^{A_pE}(1 - r_i^{A_p}) + P_i^{UE}\sigma^{A_pE}m(1 - r_i^{A_n}) \\
& + P_i^{UE}\sigma^{A_nE}(1 - m)(1 - r_i^{A_n}) + P_i^{A_nE}\sigma^{UE}(1 - m)\delta \\
& + P_i^{A_nE}\sigma^{A_pE}m\delta + P_i^{A_nE}\sigma^{A_pE}m(1 - \delta) \\
& + P_i^{A_nE}\sigma^{A_nE}(1 - m)(1 - \delta) + P_i^{A_pE}\sigma^{UE}(1 - m)\delta \\
& + P_i^{A_pE}\sigma^{A_pE}m\delta + P_i^{A_pE}\sigma^{A_pE}(1 - \delta) + \varepsilon_i(1 - \gamma)
\end{aligned} \tag{26}$$

Substituting (18) - (25) in (26) leads to

$$\sum_j \left[(\mu^2 P_i^{A_pS} + \mu P_i^{US} + P_i^{A_nS})b_{ji} - \frac{\gamma}{\lambda_0}\xi_{ji} \right] \varepsilon_j = 0 \tag{27}$$

where ξ_{ji} is the element of the identity matrix.

Let $(\mu^2 P_i^{A_pS} + \mu P_i^{US} + P_i^{A_nS})b_{ji}$ be an element of matrix H . (27) can then be rewritten as:

$$\lambda_C = \frac{\gamma}{\Lambda_{max}(H)} \tag{28}$$

where $\Lambda_{max}(H)$ is the maximum eigenvalue of matrix H , while γ refers to the likelihood of an individual recovering from the disease. From the derivation presented in (28), we can observe that the epidemic critical threshold λ_C of the two-layer coupled UA_nA_pU -

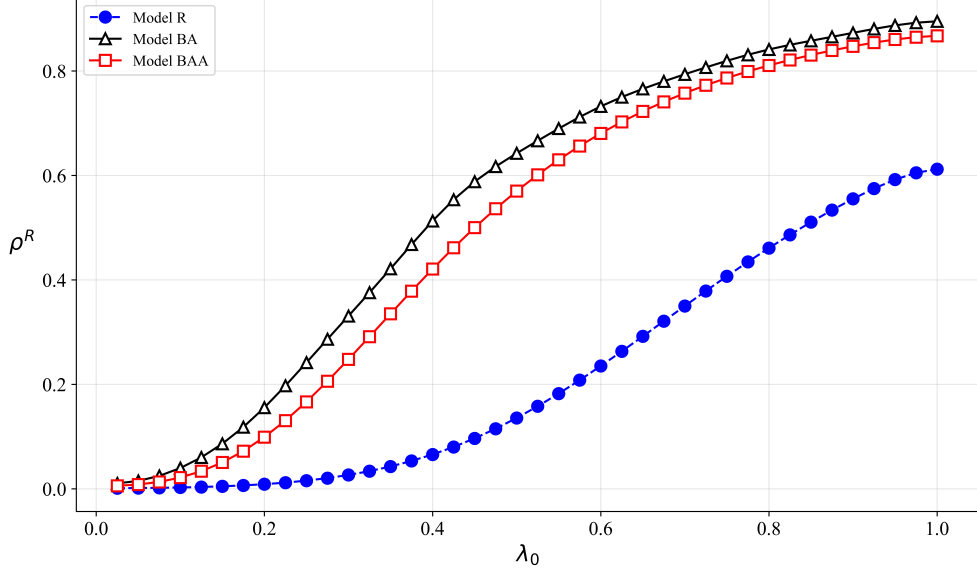


Fig. 6 Comparison of the recovered scale ρ^R for Model R, Model BA, and Model BAA under different basic disease transmission rates λ_0 . The other parameters are set as $\alpha_0 = 0.3$, $m = 0.55$, $\delta = 0.3$, $\sigma_0 = 0.45$, $\gamma = 0.65$, and $\mu = 0.7$.

SEIR model is explicitly affected by the attenuation factor μ as well as the recovery rate γ .

In subsequent experiments, we will examine the transmission dynamic of the epidemic outbreak threshold across different models as different parameters change.

4 The numerical simulations

In this section, MMCA is adopted to verify the applicability of our network model, which is derived from an age-contact matrix[21]. By adjusting various parameters, we examine the transmission dynamic of network models with different structures. The networks reconstructed from the real dataset[21] include Model BAA and Model R. In addition, a standard BA network (Model BA) without any manipulation is prepared for experimental comparison. Each of these networks contains 8000 nodes and has a mean degree $\langle k \rangle$ of approximately 6. They serve as the physical connect network that represents disease transmission in our experiments. To further match the characteristics of the information spreading layer as an online virtual network, we adopt a structure similar to that of Facebook[29, 30], with $N = 8000$ nodes and average degree $\langle k \rangle = 6$. Using this structure allows the spread of information during disease transmission to better reflect real-world conditions. All simulation results are obtained by averaging over 20 independent runs of the MMCA method. In addition, the initial fractions of nodes in states A_n , A_p , and I are each set to 0.01, distributed across age groups following the population distribution shown in Figure 2B.

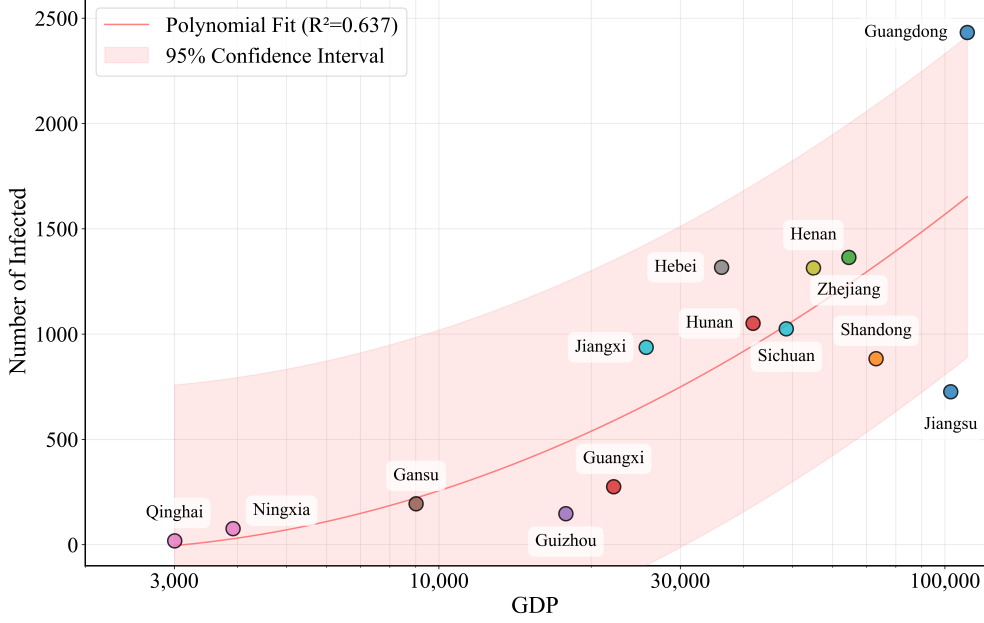


Fig. 7 Final infected population size as a function of the GDP in 2020 for 14 Chinese provinces, covering the period from 2020 to 2021. The solid line represents a polynomial fit to the simulation data, indicating the general trend of disease transmission across different GDP levels. The 2020 GDP data of each province are sourced from information released by the Chinese government, available at <https://www.tkww.hk/a/202101/30/AP6014fbf2e4b0661d556bf013.html>.

Previous studies have shown that random networks and scale-free networks exhibit different results in propagation processes due to their structural differences. As a result, they can be adapted to match various real-world transmission scenarios according to specific needs[31, 32]. For this reason, we design a set of comparative experiments to describe the differences between two distinct network structures generated from the same age dataset. As shown in Figure 6, Model R, built on a random network structure, lacks the hub nodes present in BA networks. This leads to a much bigger outbreak threshold compared to both Model BAA and Model BA, which adopt BA structures. Consequently, the disease spreads much less effectively in Model R. In addition, Model BAA modifies some edge connection patterns to make its network structure fit the age contact frequency matrix from the dataset. This operation disrupts the connection patterns between hub nodes. Hence, the outbreak threshold of Model BAA is larger than that of Model BA. This difference appears in Figure 6 as a lower recovered population density ρ^R for Model BAA than for Model BA.

To further explore how different network structures reflect real-world infection patterns, we analyzed a public dataset published on Kaggle by Chennai, Tamil Nadu, and colleagues. This dataset contains confirmed and recovered case records from January 2020 to May 2021. All data were retrieved from: <https://www.kaggle.com/datasets/>

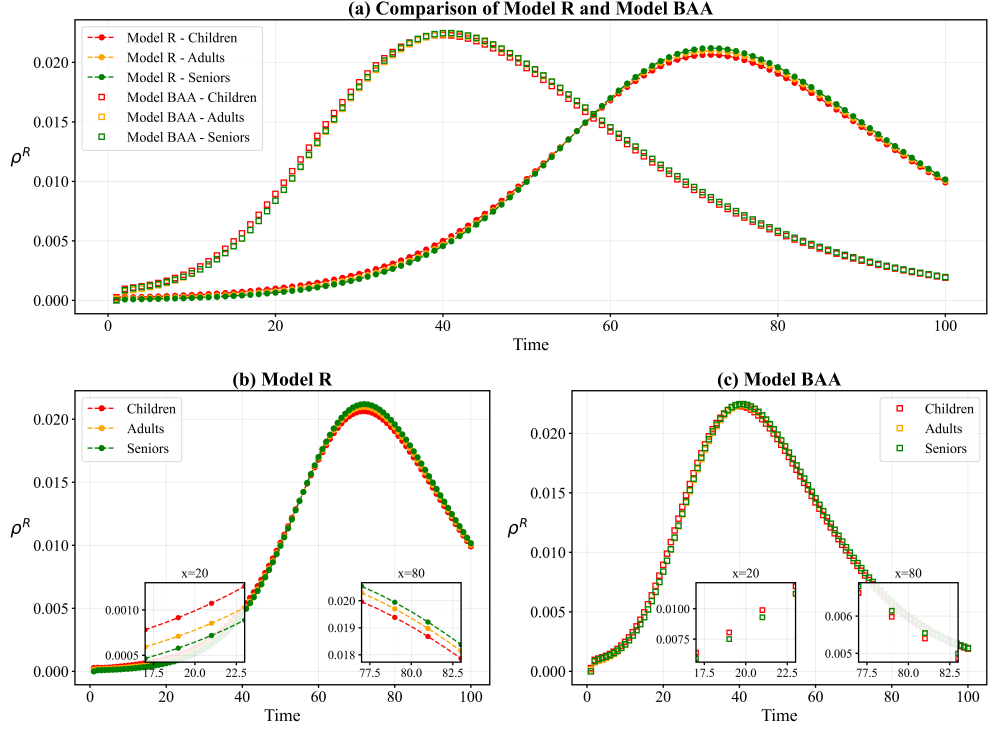


Fig. 8 (a) Comparison of the infected scale ρ^I for Model R and Model BAA over 100 time steps. (b) Infection scale ρ^I of Model R over 100 time steps t . (c) Infection scale ρ^I of Model BAA over 100 time steps t . Parameters are set as $\alpha_0 = 0.1$, $m = 0.3$, $\delta = 0.4$, $\lambda_0 = 0.8$, $\sigma_0 = 0.3$, $\gamma = 0.2$, and $\mu = 0.4$.

[sudalairajkumar/novel-corona-virus-2019-dataset](https://github.com/sudalairajkumar/novel-corona-virus-2019-dataset). As shown in Figure 7, by examining data from different economically development provinces in China, we find that provinces under varying levels of economic development can lead to the distinct final scale of epidemic diffusion. Figure 7 shows that the final scale of infection diffusion is positively correlated with the GDP of each province. In the study of disease transmission, the final scale of the epidemic is closely tied to its outbreak threshold. Specifically, regions with a smaller outbreak threshold tend to experience a larger final spread of infections. As shown in Figure 7, the final infection scale across Chinese provinces is positively correlated with regional GDP. This indicates that the outbreak threshold is mathematically correlated with a region's GDP level. Consequently, different regions exhibit different epidemic sizes after an outbreak. Motivated by the above analysis, we conclude that distinct network structures can capture the calculation of outbreak thresholds for regions with different economic development levels. Specifically, Model R, generated from a random network structure, reflects the derivation of the epidemic outbreak threshold for regions with relatively lower economic development. In contrast, the model generated from a BA network structure captures the outbreak threshold derivation for regions with relatively higher economic development.

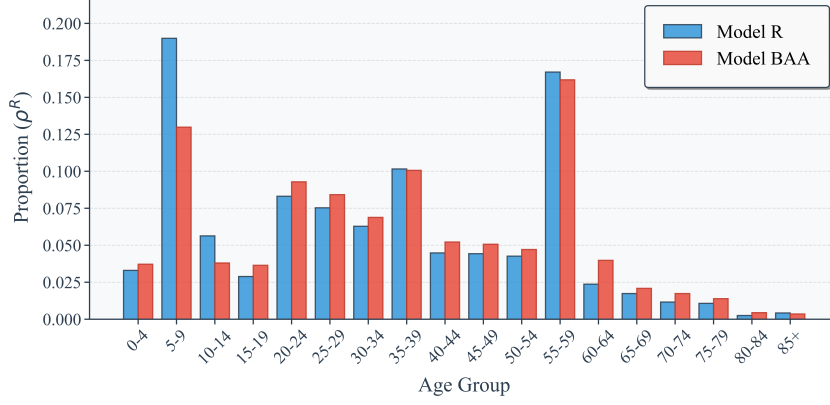


Fig. 9 Age-group proportion of recovered individuals at steady state, denoted as ρ^R . The age groups 5 – 9 and 55 – 59 exhibit a high infection scale ρ^R due to their behavioral patterns during disease transmission. Parameters are given as $\alpha_0 = 0.1, m = 0.3, \delta = 0.4, \lambda_0 = 0.5, \sigma_0 = 0.45, \gamma = 0.45$, and $\mu = 0.4$.

To further explore the validity of the above conclusion, we recorded the proportion of infected individuals ρ^I of the two models over 100 time steps. As shown in Figure 8, under the assumption that no factors interfere such as government epidemic prevention measures and the disease affects all individuals equally, the disease spreads more slowly on Model R than on Model BAA. Model R takes nearly twice as many time steps as Model BAA to reach its peak infection scale. This finding supports the conclusion that Model R and Model BAA can respectively serve as physical disease transmission models for regions at different levels of economic development. Meanwhile, we observe that in both networks, among the three age groups—children, adults, and the seniors—children initially exhibit the highest infection scale, but later become the lowest. Results obtained from a simulation where the disease affects all individuals equally indicate that, in real life, children are not only more vulnerable to infection due to weaker immunity but also face a higher risk of exposure because of their contact patterns.

To further clarify the epidemic transmission process across different age groups, the proportion of recovered individuals ρ^R for each age group is shown in Figure 9 when the system reached a steady state. Figure 9 shows that age groups 5 – 9 and 55 – 59 exhibit relatively high levels of disease spread. This result suggests that, even when the disease affects all individuals equally, people in these two age ranges still face a higher probability of infection due to their contact patterns. Therefore, among children and adults, individuals aged 5 – 9 and 55 – 59 should be considered key targets for protection.

In addition, to investigate the effect of the information spreading layer on disease transmission, three sets of experiments are presented in Figure 10. From the scale of recovered individuals ρ^R , we find that the basic probability of information transmission α_0 cannot eliminate the differences between Model R and Model BAA arising from their distinct network structures. Moreover, the outbreak threshold of Model BAA

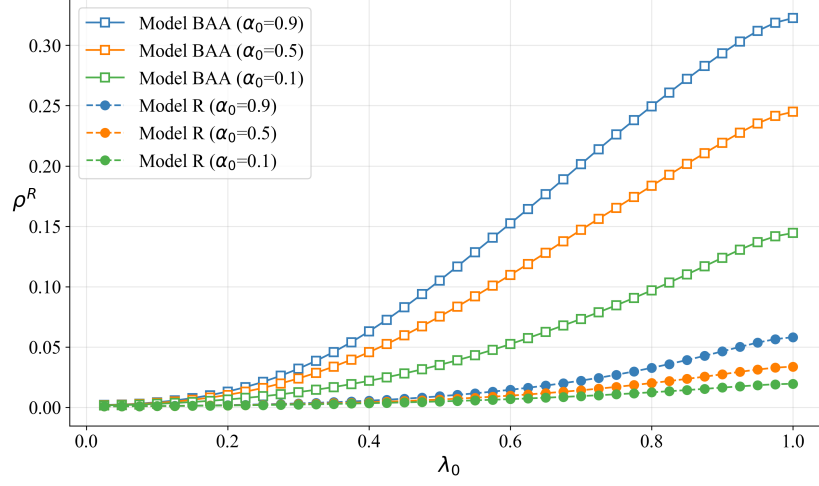


Fig. 10 Comparison of the recovered scale ρ^R between Model R and Model BAA for different basic information transmission rates α_0 and basic disease transmission rates λ_0 . The infection scale ρ^R is more affected by basic information propagation rate α_0 in Model BAA than in Model R. Parameters are set as $m = 0.3$, $\delta = 0.4$, $\sigma_0 = 0.45$, $\gamma = 0.35$, and $\mu = 0.4$.

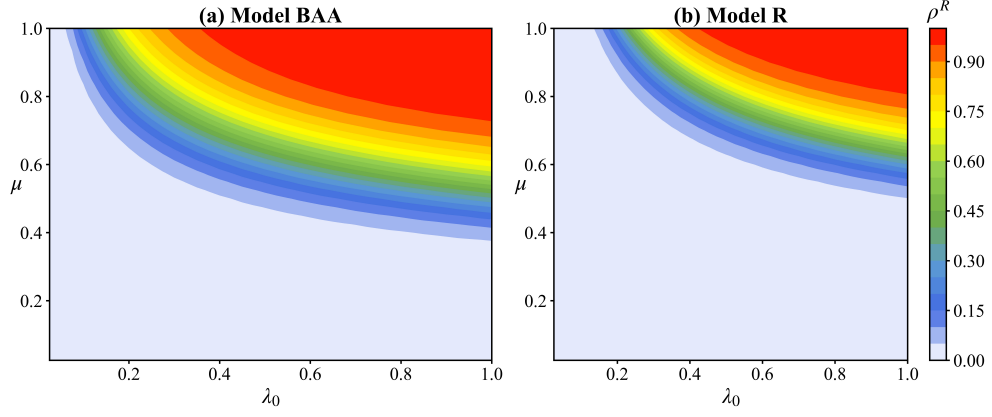


Fig. 11 Outcomes of ρ^R across different values of the baseline transmission rate λ_0 and the attenuation parameter μ . All 40×40 data points visualized in these diagrams are computed from the MMCA. Compared with Model BAA, the recovery population size ρ^R shows a stronger response to changes in the attenuation parameter μ in Model R. Parameter configurations for these simulations: $\alpha_0 = 0.45$, $m = 0.5$, $\delta = 0.2$, $\sigma_0 = 0.55$, and $\gamma = 0.6$.

remains lower than Model R across different values of α_0 . In real life, the basic probability of information transmission α_0 often reflects how effectively information flows within a region. During a disease outbreak, besides accurate prevention information,

misinformation that hinders epidemic control can also circulate. With the advancement of modern communication, promptly eliminating false information and enabling the spread of correct content over networks plays a crucial role in epidemic response.

Lastly, we can observe from [Figure 11](#) the impact of the attenuation parameter μ on the proportion of recovered individuals ρ^R . In real life, μ can be interpreted as the effectiveness of disease protection and the correctness of epidemic control measures. Higher epidemic prevention effectiveness corresponds to a smaller attenuation factor μ . Thus, as shown in [Figure 11](#), strengthening local disease protection measures can suppress the scale of disease transmission. The range of attenuation factor μ needed to curb the spread is larger for Model R than for Model BAA. Therefore, under the same level of prevention effort, disease transmission in Model R can contain the disease more quickly than Model BAA. In addition, as shown in [Figure 7](#), different network structures can reflect the mathematically derived epidemic outbreak thresholds for distinct regions. The attenuation rate μ helps explain why Model R and Model BAA can reflect the mathematical outbreak thresholds of provinces at different economic development levels.

5 Conclusion

This study developed a network reconfiguration framework that embeds age-specific contact frequency matrices into both random and BA networks, enabling a more accurate investigation of coupled information-disease transmission dynamics. Using the edge reconnecting method, we constructed two age-related network models (Model R and Model BAA), based on which we proposed a two-layer $UA_nA_pU - SEIR$ model and analyzed its propagation dynamics via the MMCA.

Our analysis, validated against real provincial epidemic data in China, yielded two key findings. First, the reconstructed networks are differentially suited to regions with varying economic development. Model R captures the transmission thresholds of less developed areas, while Model BAA aligns with those of more developed regions, explaining the empirical difference in containment effectiveness across regions. Second, we identified that children aged 5 – 9 and adults aged 55 – 59 face the highest infection risks purely due to their behavioral contact characteristics, highlighting them as priority targets for intervention.

This work provides a practical framework for constructing realistic epidemic models, but it still lacks research on time-varying networks and network structures with clustered contact environments. In addition, we did not further investigate which specific differences between BA networks and random networks enable them to reflect the outbreak thresholds derived for different regions. In future work, we will extend this framework to incorporate time-varying and setting-specific contact patterns to further improve the accuracy of dynamic epidemic predictions.

Author Contributions H. C.: Writing - original draft, Writing - review , Investigation, Methodology, Software, Visualization, Data curation. G. Y.: Validation, Supervision. H. P.: Conceptualization, Formal analysis, Funding acquisition, Project administration, Resources. All authors reviewed the manuscript.

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Data Availability This research used publicly available datasets, which are openly accessible via the official public links cited in the manuscript.

Declarations

Conflict of interest The authors declare that they have no conflict of interest.

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