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RESEARCH ARTICLE

Allocating Resources Between Asymptomatically and Symptomatically Infected Individuals on Networks

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ABSTRACT In the process of controlling infectious diseases, the investment of medical resources is essential. To address the allocation of medical resources between asymptomatically and symptomatically infected individuals, we propose a network-based SAIRS quench mean-field model. The stability of the disease-free equilibrium is proved and the condition for the existence and uniqueness of the endemic equilibrium is given with the help of Gerschgorin theorem. Numerical simulation results reveal that the fraction of the final infected population at steady state is an increasing function of the transmission rate and a decreasing function of the amount of medical resources. We also find the existence of threshold for the amount of medical resources, such that the disease can be well controlled if it is beyond the threshold. Moreover, the threshold will become larger as the transmission rate increases. Besides, the optimal resources allocation strategy is studied. When medical resources are less, allocating all to symptomatic infected individuals will minimize the fraction of the final infected population at steady state. However, with the amount of medical resources increases, a near-average distribution between asymptomatically and symptomatically and symptomatically infected individuals will result in the smallest fraction of the final infected population. Our results could have practical implications for the allocation of medical resources.

INDEX TERMS Quench mean-field model, dynamics, resources allocation strategy.

I. INTRODUCTION

In order to effectively control infectious diseases, it is essential to invest and allocate medical resources reasonably. Otherwise it will cause unnecessary waste of resources, especially during the stage of diseases outbreak, medical resources tend to be limited. Some infectious diseases (e.g. dengue fever, [1], [2] norovirus [3], [4], [5], [6] and COVID-19 [7], [8], [9] etc.) present asymptomatic infected individuals (who have no symptoms, but also infectious), which can also threaten people's health. The results of these works suggested that asymptomatic infected individuals should not be ignored. For the purpose of minimizing the fraction of infection, how to allocate medical resources between asymptomatic and symptomatic infected individuals has become a problem that needs to be solved. And this is exactly the issue will be addressed in this paper.

The amount of medical resources invested plays an important factor in the spread and control of infectious diseases. L.Böttcher et al. established a model which the recovery of sick individuals depends on the availability of resources generated by the healthy population. It was found that if the cost of recovery is higher than a critical cost, the epidemic would get out of control [10]. Chen et al. found

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that if the amount of invested resources is above a critical value, the disease can be effectively contained [11]. Based on a SIS model, Chen et al. solved the optimal medical resources allocation problem to reduce the prevalence of disease. It was proved that the outbreak of the epidemic will be maximally suppressed when the curing rate of each node is proportional to its degree [12]. Li et al. proposed a two-epidemic spreading model under the control of public resources. The resource thresholds of the two diseases and optimal allocation coefficients were obtained. They found that when the resources are limited, preferentially control the disease with lower transmission rate is a better strategy [13]. The optimal resources allocation strategy was found under convex framework to control the spread of epidemic outbreak in a given network [14], [15], [16]. Wang et al. studied optimal quarantine measures from the perspective of optimal control, which provides a broader idea for the allocation of medical resources [17]. Optimal vaccination and treatment strategies for a novel SIRS model with time delay are investigated in [18].

In addition, Jiang et al. studied a two-layer network model which considers a variable recovery rate related to resource. The results showed that when the amount of resource beyond a threshold, the disease may be effectively eradicated and layer-layer connection strength can transform the type of phase transition [19]. A two-layer network model with the interaction between individual resource support and disease transmission was studied in [20]. The recovery of infected individuals depends on the resources received from healthy neighbors and the results showed that there existed hybrid phase transition and hysteresis loop. Sun et al. proposed a multilayer network model to study the impact of resource diffusion on disease transmission in higher-order networks. It was found that increasing the diffusion of resources on 2-simplexes can contain the spread and outbreak of the epidemic [21]. Huang et al. proposed a coupled resource-epidemic model on a time-varying multiplex network. The results showed that the stronger the heterogeneity of activity and the greater contact capacity of individuals in the resource layer can promote the resource diffusion to a greater extent and effectively inhibit the spread of the epidemic [22]. Despite many results on the allocation of medical resources, few people studied how to allocate medical resources between asymptomatic and symptomatic infected individuals.

When complex networks were used to study infectious diseases, the impact of structural characteristics of individual contact networks on diseases transmission began to be considered. Some scholars improved the heterogeneous mean-field method and proposed quench mean-field method [23], [24], [25]. This method not only considers the heterogeneity of individuals, but also the topological structure of the network using adjacency matrix. Here are some literatures on the analysis of infectious diseases by establishing quench mean-field models. Yang et al. constructed a epidemic control synchronization model with

the inhibition of contact behavior and obtained the epidemic threshold [26]. Liu et al. combined the typical SIS model with behavioral game to analyze the influence of behavior on infectious diseases on complex networks [27]. Recently, Zhang et al. proposed a discrete-time model combining infectious diseases and the game of non-drug interventions. The epidemic threshold and the optimal control probability of infected individuals were obtained [28].

Robinson and Stilianakis [29] analyzed the dynamics of SAIRS epidemic model with the emergence of drug resistance in the presence of asymptomatic infections. The effect of the emergence of resistance after treatment on the system dynamics was discussed. The global stability of SAIRS compartmental model with vaccination was studied in [30]. The expression of the basic reproduction number R_0 was given and it was proved that the disease-free equilibrium is globally asymptotically stable if $R_0 < 1$. When $R_0 > 1$, the endemic equilibrium is globally asymptotically stable. Similar to these two literatures, the population is divided into the same four states and asymptomatic infected individuals are also considered. The difference is that we consider a recovery rate function which depends on the amount of medical resources, and discuss the problem of medical resources allocation. We find that a critical resource amount is needed to eliminate the disease in this paper. Besides, there exist different optimal resources allocation coefficients for different amounts of medical resources. The main contributions of this paper are as follows:

• A new quench mean-field SAIRS model related to the amount of medical resources and the coefficient of resource allocation is proposed;

• Due to the high dimension and complexity of the model, it is also a breakthrough to prove the stability of disease-free equilibrium and the existence of endemic equilibrium theoretically;

• From the perspective of numerical simulations, it explores how to allocate medical resources between asymptomatically and symptomatically infected individuals to minimize the final infected population, which is an innovation of this paper.

The structure of this paper is as follows. In Section II, a new network-based SAIRS epidemic model is proposed. The existence and stability of the disease-free and endemic equilibrium are studied in Section III. The numerical simulations are performed in Section IV. Lastly, the conclusions and discussions are presented in Section V.

II. MODEL DESCRIPTION

The striking features of the recent COVID-19 pandemic is the presence of asymptomatic infected and the repeated infection of people [30]. Moreover, the propagation law is related to the network structure (the connection relationship between people). Based on these characteristics, we propose a quench mean-field SAIRS model in which both the asymptomatically infected and symptomatically infected individuals are infectious in this section. Specifically, this



FIGURE 1. Flow chart of disease transmission.

model considers a physical contact network consisting of N nodes, each node represents an individual. The adjacency matrix of the network is represented by $B = (b_{ij})_{N \times N}$. If individual *i* has contact with individual *j*, then $b_{ij} = 1$, and if not, then $b_{ij} = 0$. The diagonal elements of matrix *B* is $b_{ii} = 0$. Each node is assumed to have one of four states: susceptible (*S*), asymptomatically infected (*A*), symptomatically infected (*I*) or recovered (*R*).

As shown in Figure 1, some susceptible people become asymptomatic after being infected, some will show symptoms, and part of the asymptomatic infected people will also show symptoms over time. Infected people will eventually recover with the help of medical resources. The recovery rate of an individual is positively related to the amount of medical resources invested [8], [9]. Inspired mainly by the work of reference [10], we set the recovery rate of asymptomatic and symptomatic infected individuals as $e^{-\frac{1}{wM}}$ and $e^{-\frac{1}{(1-w)M}}$ respectively, in which *M* is the total amount of medical resources invested and *w* is the resource allocation coefficient used to adjust the allocation of total resources between *A* and *I*. Because of the loss of immune function, the recovered individuals have the possibility of being re-infected, that is, becoming susceptible again.

Denote $S_i(t)$, $A_i(t)$, $I_i(t)$, $R_i(t)$ be the probabilities associated with node *i* being in the states *S*, *A*, *I* or *R* at time *t* respectively. Then, the network-based SAIRS quench mean-field model is described as follows and the interpretation of the other parameters are shown in Table 1.

$$\begin{cases} \dot{S}_{i}(t) = -S_{i}(t)P_{i}(t) + \delta R_{i}(t), \\ \dot{A}_{i}(t) = \epsilon S_{i}(t)P_{i}(t) - qA_{i}(t) - e^{-\frac{1}{wM}}A_{i}(t), \\ \dot{I}_{i}(t) = (1 - \epsilon)S_{i}(t)P_{i}(t) + qA_{i}(t) - e^{-\frac{1}{(1 - w)M}}I_{i}(t), \\ \dot{R}_{i}(t) = e^{-\frac{1}{wM}}A_{i}(t) + e^{-\frac{1}{(1 - w)M}}I_{i}(t) - \delta R_{i}(t), \end{cases}$$
(1)

where

$$P_i(t) = 1 - \prod_j [1 - b_{ij}(\beta_1 A_j(t) + \beta_2 I_j(t))], \qquad (2)$$

is the probability that node i is infected by at least a infected neighbor. The infected individuals can pass the disease to their susceptible neighbors until they finally recover.

Remark 1: It is easy to find that $S_i(t) + A_i(t) + I_i(t) + R_i(t) = 1, S_i(t), A_i(t), I_i(t), R_i(t) \ge 0$, for all $i \in \{1, 2, \dots, N\}$.

TABLE 1. Definition of parameters.

Parameter	Definition
β_1	Transmission rate of asymptomatically infected individuals
β_2	Transmission rate of symptomatically infected individuals
δ	Transmission rate from R to S
ϵ	Rate of being asymptomatic after infected
q	Transmission rate from A to I

III. EXISTENCE AND STABILITY OF EQUILIBRIUM

The existence and stability of equilibrium will be proved in this section. First, we present a proof of the stability of the disease-free equilibrium.

A. STABILITY OF THE DISEASE-FREE EQUILIBRIUM

It is easy to calculate the unique disease-free equilibrium given by $E_0 = (\underbrace{1, 1, \dots, 1}_{N}, \underbrace{0, 0, \dots, 0}_{3N}) \in \mathbb{R}^{4N}$, when the right side of system (1) to be equal to zero and $A_i(t) = I_i(t) = 0$, that is, the infectious disease will eventually

become extinct. Since $S_i(t) = 1 - A_i(t) - I_i(t) - R_i(t)$, so

we can only study the following system:

$$\begin{cases}
\dot{A}_{i}(t) = \epsilon(1 - A_{i}(t) - I_{i}(t) - R_{i}(t))P_{i}(t) - qA_{i}(t) \\
-e^{-\frac{1}{wM}}A_{i}(t), \\
\dot{I}_{i}(t) = (1 - \epsilon)(1 - A_{i}(t) - I_{i}(t) - R_{i}(t))P_{i}(t) \\
+qA_{i}(t) - e^{-\frac{1}{(1 - w)M}}I_{i}(t), \\
\dot{R}_{i}(t) = e^{-\frac{1}{wM}}A_{i}(t) + e^{-\frac{1}{(1 - w)M}}I_{i}(t) - \delta R_{i}(t).
\end{cases}$$
(3)

Furthermore, the Jacobian matrix of system (3) at E_0 is

$$J = \begin{pmatrix} \epsilon \beta_1 B - (q + e^{-\frac{1}{wM}})E & \epsilon \beta_2 B & O \\ (1 - \epsilon)\beta_1 B + qE & (1 - \epsilon)\beta_2 B - e^{-\frac{1}{(1 - w)M}}E & O \\ e^{-\frac{1}{wM}}E & e^{-\frac{1}{(1 - w)M}}E & -\delta E \end{pmatrix},$$

where *E* represents the $N \times N$ unit matrix and *O* represents the $N \times N$ zero matrix.

Theorem 1: If $\rho(B) < c$, the disease-free equilibrium E_0 of system (3) is locally asymptotically stable, where $\rho(B)$ is the spectral radius of the adjacency matrix B and

$$c = \frac{(q + e^{-\frac{1}{wM}})e^{-\frac{1}{(1-w)M}}}{\epsilon\beta_1 e^{-\frac{1}{(1-w)M}} + \beta_2(q + e^{-\frac{1}{wM}} - \epsilon e^{-\frac{1}{wM}})}$$

Proof: The local stability of E_0 depends on the sign of the real parts of the eigenvalues of the Jacobian matrix J. The eigenvalues of matrix J are obtained from the characteristic equation $\tau_1(\lambda) = \det(J - \lambda E) = \det[(-\delta - \lambda)E] \cdot \det\left[(q + e^{-\frac{1}{wM}} + \lambda)(e^{-\frac{1}{(1-w)M}} + \lambda)E - (\epsilon\beta_1(e^{-\frac{1}{(1-w)M}} + \lambda) + (1 - \epsilon)\beta_2(q + e^{-\frac{1}{wM}} + \lambda) + q\epsilon\beta_2)B\right] = 0$. Since the eigenvalues do not change if one multiplies the characteristic polynomial with a constant. Therefore, a more convenient form of the second part of characteristic polynomial is $\tau_2(\lambda) = \det\left[\frac{(q+e^{-\frac{1}{wM}} + \lambda)(e^{-\frac{1}{(1-w)M}} + \lambda)}{\epsilon\beta_1(e^{-\frac{1}{(1-w)M}} + \lambda)(e^{-\frac{1}{(1-w)M}} + \lambda)}E_1 - B\right]$.

Let Λ is the eigenvalue of the adjacency matrix *B*, then

$$\frac{\left(q+e^{-\frac{1}{wM}}+\lambda\right)(e^{-\frac{1}{(1-w)M}}+\lambda)}{\epsilon\beta_1(e^{-\frac{1}{(1-w)M}}+\lambda)+(1-\epsilon)\beta_2\left(q+e^{-\frac{1}{wM}}+\lambda\right)+q\epsilon\beta_2}=\Lambda.$$

By simple calculation, the following equation can be obtained

$$\lambda^2 + a_1\lambda + a_0 = 0,$$

where $a_1 = q + e^{-\frac{1}{wM}} + e^{-\frac{1}{(1-w)M}} - \epsilon\beta_1\Lambda - (1 - \epsilon)\beta_2\Lambda$, $a_0 = (q + e^{-\frac{1}{wM}})e^{-\frac{1}{(1-w)M}} - \epsilon\beta_1e^{-\frac{1}{(1-w)M}}\Lambda - \beta_2\Lambda(q+e^{-\frac{1}{wM}}-\epsilon e^{-\frac{1}{wM}})$. According to the theory of quadratic equations, if the two inequalities $a_1 > 0$, $a_0 > 0$ hold simultaneously, the eigenvalues of the Jacobian matrix will be less than 0, and hence the disease-free equilibrium E_0 is locally asymptotically stable. Specifically, we can obtain the following results by solving the inequalities

$$\begin{cases} \Lambda < \frac{q + e^{-\frac{1}{wM}} + e^{-\frac{1}{(1-w)M}}}{\epsilon\beta_1 + (1-\epsilon)\beta_2}, \\ \Lambda < \frac{(q + e^{-\frac{1}{wM}})e^{-\frac{1}{(1-w)M}}}{\epsilon\beta_1 e^{-\frac{1}{(1-w)M}} + \beta_2(q + e^{-\frac{1}{wM}} - \epsilon e^{-\frac{1}{wM}})}. \end{cases}$$
(4)

Since the largest eigenvalue of the adjacency matrix is the spectral radius, and it is easy to verify that $\frac{q+e^{-\frac{1}{wM}}+e^{-\frac{1}{(1-w)M}}}{\epsilon\beta_1+(1-\epsilon)\beta_2} > \frac{(q+e^{-\frac{1}{wM}})e^{-\frac{1}{(1-w)M}}}{\epsilon\beta_1e^{-\frac{1}{(1-w)M}}+\beta_2(q+e^{-\frac{1}{wM}}-\epsilon e^{-\frac{1}{wM}})}.$ Therefore, if $\rho(B) < c$, then E_0 is locally asymptotically stable.

Remark 2: In order to control infectious diseases, the value of *c* should be increased as much as possible. As shown in Figure 2, *c* is a monotonically decreasing function of $\beta_1, \beta_2, \epsilon$ and monotonically increasing function of *q*, *M*. Therefore, we should take some measures to reduce the transmission rate, such as wearing masks, isolating infected people, vaccinating and increasing the investment of medical resources. With the increase of *w*, *c* first increases and then decreases. When the value of *w* is around 0.5, *c* reaches the maximum value, that is, it is most conducive to suppress the spread of infectious diseases, which is consistent with the results reflected in Figure 7.

Remark 3: Due to the complexity of c, it is difficult to obtain the theoretical expression of the critical amount of medical resources, but it exists, as shown in Figure 5.

B. EXISTENCE OF THE ENDEMIC EQUILIBRIUM

In this subsection, we will give the condition for the existence and uniqueness of the positive equilibrium. When there exist endemic equilibrium, it means that the infectious disease will become endemic, that is, persist for a long time. In controlling infectious diseases, we should try to eliminate the disease, and if not possible, we should actively take measures to reduce the fraction of the infected population as much as possible.

Lemma 1 (Gerschgorin Theorem, Ref. [31]): Let $B = (b_{ij}) \in C^{n \times n}$ and let $r_i = \sum_{j=1, j \neq i}^n |b_{ij}|, i = 1, 2, \dots, n$. Then, all the eigenvalues of B lie in the union of n closed Theorem 2: When $\rho(B) > c$, system (1) admits a unique endemic equilibrium.

Proof: Let the right side of system (1) to be equal to zero, we can obtain

$$\begin{cases} -S_i P_i + \delta R_i = 0, \\ \epsilon S_i P_i - q A_i - e^{-\frac{1}{wM}} A_i = 0, \\ (1 - \epsilon) S_i P_i + q A_i - e^{-\frac{1}{(1 - w)M}} I_i = 0, \\ e^{-\frac{1}{wM}} A_i + e^{-\frac{1}{(1 - w)M}} I_i - \delta R_i = 0. \end{cases}$$

Then we can get $A_i = \frac{\epsilon S_i P_i}{q + e^{-\frac{1}{wM}}}$, $I_i = \frac{qS_i P_i + (1-\epsilon)S_i P_i e^{-\frac{1}{wM}}}{(q + e^{-\frac{1}{wM}})e^{-\frac{1}{(1-w)M}}}$, $R_i = \frac{S_i P_i}{\delta}$. Substituting them into equation $S_i + A_i + I_i + R_i = 1$ yields $S_i = \frac{\delta(q + e^{-\frac{1}{wM}})}{L_1}$, where $L_1 = (P_i + \delta)(q + e^{-\frac{1}{wM}}) + \delta P_i(q + e^{-\frac{1}{wM}} - \epsilon e^{-\frac{1}{wM}})e^{\frac{1}{(1-w)M}} + \epsilon \delta P_i$. Furthermore, it can be obtained that $A_i = \frac{\epsilon \delta P_i}{L_1}$, $I_i = \frac{\delta P_i(q + e^{-\frac{1}{wM}} - \epsilon e^{-\frac{1}{wM}})e^{\frac{1}{(1-w)M}}}{L_1}$. Since $P_i = 1 - \prod_j [1 - b_{ij}(\beta_1 A_j + \beta_2 I_j)] \approx \sum_j b_{ij}(\beta_1 A_j + \beta_2 I_j)$, then a self-consistency equation can be obtained as follows: $P_i = \sum_j b_{ij} \frac{L_2 P_i}{L_1}$, where $L_2 = \epsilon \delta \beta_1 + \delta(q + e^{-\frac{1}{wM}} - \epsilon e^{-\frac{1}{wM}})\beta_2 e^{\frac{1}{(1-w)M}}$. We note the function

$$F(P_i) = \sum_{j} b_{ij} \frac{L_2 P_i}{L_1}.$$
 (5)

It is easy to find that $P_i = 0$ is always a solution of equation (5), that is to say model (1) has a disease-free equilibrium. In addition,

$$F'(P_i) = \sum_{j} b_{ij} \frac{\delta(q + e^{-\frac{1}{wM}})L_2}{L_1^2} > 0,$$

$$F''(P_i) = \sum_{j} b_{ij} \delta L_2 (q + e^{-\frac{1}{wM}}) \frac{-2}{L_1^3} < 0.$$

So, $F(P_i)$ is a monotonically increasing and concave function. Therefore, a nontrivial solution exists only if

$$F'(P_i)|_{P_i=0} = \sum_j b_{ij} \frac{L_2}{\delta(q+e^{-\frac{1}{wM}})} > 1 \Leftrightarrow c < \sum_j b_{ij}.$$

According to Lemma 1, we can conclude that $\rho(B) \le \sum_j b_{ij}$. Therefore, to sum up, when $\rho(B) > c$, system (1) has a unique endemic equilibrium.

IV. SIMULATIONS

Due to the difficulty of constructing a real infectious disease transmission network, the numerical simulations section of this paper is simulated on the BA scale-free network. BA scale-free network is a kind of complex networks whose degree distribution obey the power law distribution. First, we construct a scale-free network with 1000 nodes. The specific method is as follows: at the beginning, there are



FIGURE 2. The relationship between *c* and each parameter. The value of the parameters are $\beta_1 = 0.06$, $\beta_2 = 0.01$, $\epsilon = q = w = 0.5$ and M = 10.

five nodes in the network, then each time a new node is added and connected to an existing node according to a preferential connection rule. In other words, the nodes with greater degree are more likely to be selected and connected, and the more connected edges are accumulated with the generation of the network. So the adjacency matrix *B* is determined based on the generated network and $\rho(B) =$ 19.9044. We define the fraction of the infected population as $I(t) = \frac{1}{1000} \sum_{i=1}^{1000} (A_i(t) + I_i(t))$ and denote *I* be the fraction of the final infected population at steady state in our paper.

A. THE DYNAMIC BEHAVIOR

We select the parameter $\delta = 0.1$ and the values of other parameters are the same as those in Figure 2. In this case, Figure 3(a): M = 10, we can calculate $c = 32.0804 > \rho(B)$, so E_0 is locally asymptotically stable from the result of Theorem 1. Observing the time evolution of the fraction of the infected population with 10 different initial conditions, we can see that $\lim_{t\to\infty} I(t) = 0$. This is consistent with the theoretical result. In Figure 3(b), just decreasing the value M = 0.8, then $c = 6.0689 < \rho(B)$, and then endemic equilibrium may be stable. However, due to the difficulty, the



FIGURE 3. The time evolution of the fraction of the infected population with 10 different initial conditions.



FIGURE 4. (a) Dependence of *I* on β_1 for different values of *M*, where $\beta_2 = 0.01$; (b) Dependence of *I* on β_2 for different values of *M*, where $\beta_1 = 0.06$. The other parameter values are the same as those in Figure 3.

theoretical proof of the stability of the endemic equilibrium is not given in this paper. Therefore, the amount of medical resources affects the dynamics of system (1) and plays an important role in controlling infectious diseases.

B. A CRITICAL RESOURCES AMOUNT FOR EPIDEMIC CONTROL

As can be seen from Figure 4(a), *I* is a monotone increasing function of β_1 , which is consistent with the law of the spread of infectious diseases. When M = 0.8, even if β_1 is small, the infectious disease will persist. When enough medical resources are invested, there exists a critical value of β_1 , that is, the system will undergo a transition from no disease to endemic disease. Therefore, in the process of controlling infectious disease, adequate medical resources must be invested even if the transmission rate is low.

The results reflected in Figure 4(b) are roughly similar to those in Figure 4(a), hence the difference is only explained here. When M = 0.8, *I* rapidly increases to a larger value as β_2 gradually increases. So, enough medical resources should be invested as early as possible to hinder the spread of infectious diseases, while β_2 is still small, otherwise the epidemic will soon break out. In order to further study the impact of medical resources on diseases transmission, we present the graphs of the fraction of the final infected population at steady state as a function of M by numerical iteration. As can be seen from Figure 5(a), there exists a critical value of M in the system, denoted as M_c . When $M < M_c$, the disease will persist, and I decreases as M increases. If $M > M_c$, the disease can be well contained. Moreover, M_c will be bigger with the larger β_1 . This means that when the transmission rate of asymptomatically infected individuals increases, more medical resources need to be invested in order to better control infectious diseases.

Similar results can be found in Figure 5(b), I is also decreasing with the increase of M. Besides, if β_2 increases, it also requires more medical resources to mitigate the spread of the diseases. Therefore, the transmission rate of asymptomatically and symptomatically infected individuals should be reduced as much as possible, such as wearing masks, self-isolation, vaccination and so on. In this way, it facilitates the control of infectious diseases.

The joint influence of β_1 and M on the fraction of the final infected population at steady state are reflected in Figure 6(a). It is easy to find that when β_1 increases, I also increases, while I decreases as M increases. This



FIGURE 5. Influence of medical resources amount on the fraction of the final infected population at steady state: (a) dependence of *I* on *M* for different values of β_1 , where $\beta_2 = 0.01$; (b) dependence of *I* on *M* for different values of β_2 , where $\beta_1 = 0.06$. The other parameter values are the same as those in Figure 3.



FIGURE 6. (a) The joint influence of β_1 and *M* on the fraction of the final infected population at steady state, where $\beta_2 = 0.03$; (b) The joint influence of β_2 and *M* on the fraction of the final infected population at steady state, where $\beta_1 = 0.06$. The other parameter values are the same as those in Figure 3.

result is consistent with those reflected in Figure 4 and Figure 5. We also observe that M has a greater influence on I, that is, when β_1 is fixed, the variation of I is larger as M changes. Therefore, when infectious diseases occur, the investment of medical resources is essential. The result reflected in Figure 6(b) is basically similar to that in Figure 6(a), except that when β_2 is small enough, as long as *M* increases slightly, *I* will reach a lower value quickly, which is favorable for the control of infectious diseases. This also gives us an inspiration: we should take measures to reduce the transmission rate as much as possible, such as: extensive media coverage, minimizing gathering behavior, strengthening physical exercise to improve immunity, etc. In this way, through the investment of medical resources, infectious diseases are relatively easy to be controlled, and the possibility of large-scale outbreak will be greatly reduced.

C. OPTIMAL RESOURCES ALLOCATION STRATEGY

To investigate the joint effect of w and M on the fraction of the final infected population at steady state, Figure 7(a)

is given. We can observe that the larger M is, I will be smaller regardless of the value of w. When M is fixed, the change of I is complex with the increase of w. As shown in Figure 7(b), when M = 3, I first increases, then decreases and then increases, and reaches the minimum when w = 0.4. Obviously, we can see that when the amount of medical resources is small, I will reach the minimum if all resources are allocated to symptomatic infected individuals (i.e., w = 0). This can be explained that most infected people with symptoms will seek medical help (such as medicine, masks, hospitalization, etc.) in order to recover as soon as possible. Asymptomatic infected individuals do not know they have the virus and do not feel sick, so they will not take any medical help.

However, as M increases gradually, the horizontal position of the red dots will shift to the right, gradually approach 0.5. This indicates that if there are sufficient medical resources available, allocating them nearly equally between asymptomatically and symptomatically infected individuals will minimize I. This is because asymptomatic infected individuals are also infectious, with a probability q of



FIGURE 7. The parameter values are $\beta_1 = 0.06$, $\beta_2 = 0.03$ and the other parameter values are the same as those in Figure 3. (a) The joint influence of w and M on the fraction of the final infected population at steady state, where the red dots represents the value of w that makes I to be the minimum value for fixed M; (b) The influence of w on the fraction of the final infected population at steady state for M = 3.

transitioning from A to I. Therefore, when medical resources are adequate, a portion of these resources should be allocated to asymptomatic infected individuals.

V. CONCLUSION AND DISCUSSIONS

A quench mean-field SAIRS model with recovery rate dependent on medical resources is established in this paper. We analyzed its dynamic properties and studied the influence of both transmission rate and the amount of medical resources on the fraction of the final infected population at steady state. The numerical simulation results showed that *I* is an increasing function of β_1 (or β_2) and a decreasing function of *M*. We found that there is a critical value of medical resources in the system. When enough medical resources are invested, infectious diseases will be timely controlled.

In addition, the joint impact of the amount of medical resources and resources allocation coefficient on I was researched. As M increases gradually, a distribution that is roughly equal between asymptomatic and symptomatic infected individuals will yield the smallest fraction of the final infected population. The results suggested that the asymptomatic infected individuals should also be paid attention in the process of controlling infectious diseases.

The SAIRS model is applicable for some diseases which appear asymptomatic infected individuals and the recovered individuals may be infected again, such as norovirus and COVID-19. The medical resources allocation strategy discussed in this paper is suitable for these diseases and has certain reference value for other diseases. When infectious diseases occur, it is necessary to invest certain amount of medical resources. If the amount of resources invested exceeds a critical value, the epidemic can be completely controlled. When there is a shortage of medical resources, more people will become infected, and giving priority to symptomatically infected will minimize the fraction of the final infected population. As the amount of resources increases, nearly equal allocation between asymptomatically and symptomatically infected individuals will minimize the fraction of the final infected population. In this way, the probability of large-scale outbreak of infectious diseases is reduced. Of course, in addition to the investment of medical resources, some measures can also be taken actively, such as: the use of media to educate people to wear masks, take the initiative to self-isolate, vaccination and so on, these are also conducive to the control of infectious diseases.

In a more realistic case, due to the evolution of infectious diseases, economic, region and other factors, the amount of medical resources invested tends to be dynamically adjusted and changed. However, the treatment of medical resources in system (1) is undoubtedly a simplification. In the real world, the spread of one pathogen may be affected by the presence of other pathogens, that is, two infectious diseases may spread at the same time [32]. So, how to rationally allocate medical resources so that both infectious diseases can be effectively controlled will be a topic worth studying. In addition, we did not consider specific infectious disease, and should use real data to analyze the impact of medical resources on disease transmission. For example, the impact of two major events on the transmission trend of COVID-19 was discussed in the reference [33]. We will consider these issues in the future.

REFERENCES

- M. Grunnill, "An exploration of the role of asymptomatic infections in the epidemiology of dengue viruses through susceptible, asymptomatic, infected and recovered (SAIR) models," *J. Theor. Biol.*, vol. 439, pp. 195–204, Feb. 2018.
- [2] A. Mishra and S. Gakkhar, "Modeling of dengue with impact of asymptomatic infection and ADE factor," *Differ. Equ. Dyn. Syst.*, vol. 28, no. 3, pp. 745–761, Jul. 2020.
- [3] K. Y. Leung, P. Trapman, and T. Britton, "Who is the infector? Epidemic models with symptomatic and asymptomatic cases," *Math. Biosci.*, vol. 301, pp. 190–198, Jul. 2018.
- [4] C. I. Gallimore, D. Cubitt, N. du Plessis, and J. J. Gray, "Asymptomatic and symptomatic excretion of noroviruses during a hospital outbreak of gastroenteritis," *J. Clin. Microbiol.*, vol. 42, no. 5, pp. 2271–2274, May 2004.

- [5] C. Garciia, H. L. DuPont, K. Z. Long, J. I. Santos, and G. Ko, "Asymptomatic norovirus infection in Mexican children," J. Clin. Microbiol., vol. 44, no. 8, pp. 2997–3000, Aug. 2006.
- [6] F. Bucardo, J. Nordgren, B. Carlsson, E. Kindberg, M. Paniagua, R. Möllby, and L. Svensson, "Asymptomatic norovirus infections in nicaraguan children and its association with viral properties and histoblood group antigens," *Pediatric Infectious Disease J.*, vol. 29, no. 10, pp. 934–939, 2010.
- [7] M. Rafiq, J. E. Macías-Díaz, A. Raza, and N. Ahmed, "Design of a nonlinear model for the propagation of COVID-19 and its efficient nonstandard computational implementation," *Appl. Math. Model.*, vol. 89, pp. 1835–1846, Jan. 2021.
- [8] S. M. Garba, J. M.-S. Lubuma, and B. Tsanou, "Modeling the transmission dynamics of the COVID-19 pandemic in south Africa," *Math. Biosci.*, vol. 328, Oct. 2020, Art. no. 108441.
- [9] A. Mahajan, N. A. Sivadas, and R. Solanki, "An epidemic model SIPHERD and its application for prediction of the spread of COVID-19 infection in India," *Chaos, Solitons Fractals*, vol. 140, Nov. 2020, Art. no. 110156.
- [10] L. Böttcher, O. Woolley-Meza, N. A. M. Araújo, H. J. Herrmann, and D. Helbing, "Disease-induced resource constraints can trigger explosive epidemics," *Sci. Rep.*, vol. 5, no. 1, Nov. 2015, Art. no. 16571.
- [11] X. Chen, T. Zhou, L. Feng, J. Liang, F. Liljeros, S. Havlin, and Y. Hu, "Non-trivial resource amount requirement in the early stage for containing fatal diseases," 2016, arXiv:1611.00212.
- [12] H. Chen, G. Li, H. Zhang, and Z. Hou, "Optimal allocation of resources for suppressing epidemic spreading on networks," *Phys. Rev. E*, vol. 96, no. 1, Jul. 2017, Art. no. 012321.
- [13] J. Li, C. Yang, C. Fu, Y. Gao, and H. Yang, "Cooperative epidemics spreading under resource control," *Chaos*, vol. 28, no. 11, Nov. 2018, Art. no. 113116.
- [14] C. Enyioha, A. Jadbabaie, V. Preciado, and G. Pappas, "Distributed resource allocation for control of spreading processes," in *Proc. Eur. Control Conf. (ECC)*, Jul. 2015, pp. 2216–2221.
- [15] V. M. Preciado, M. Zargham, C. Enyioha, A. Jadbabaie, and G. Pappas, "Optimal vaccine allocation to control epidemic outbreaks in arbitrary networks," in *Proc. 52nd IEEE Conf. Decis. Control*, Dec. 2013, pp. 7486–7491.
- [16] C. Nowzari, V. Preciado, and G. Pappas, "Optimal resource allocation for control of networked epidemic models," *IEEE Trans. Control Netw. Syst.*, vol. 4, no. 2, pp. 159–169, Sep. 2017.
- [17] Y. Wang, S. Chen, D. Yu, L. Liu, and K.-K. Shang, "Propagation dynamics of an epidemic model with heterogeneous control strategies on complex networks," *Symmetry*, vol. 16, no. 2, p. 166, Jan. 2024.
- [18] L. Liu, S. Chen, M. Small, J. M. Moore, and K. Shang, "Global stability and optimal control of epidemics in heterogeneously structured populations exhibiting adaptive behavior," *Commun. Nonlinear Sci. Numer. Simul.*, vol. 126, Nov. 2023, Art. no. 107500.
- [19] J. Jiang and T. Zhou, "Resource control of epidemic spreading through a multilayer network," *Sci. Rep.*, vol. 8, no. 1, Jan. 2018, Art. no. 1629.
- [20] X. Chen, R. Wang, M. Tang, S. Cai, H. E. Stanley, and L. A. Braunstein, "Suppressing epidemic spreading in multiplex networks with socialsupport," *New J. Phys.*, vol. 20, no. 1, Jan. 2018, Art. no. 013007.
- [21] Q. Sun, Z. Wang, D. Zhao, C. Xia, and M. Perc, "Diffusion of resources and their impact on epidemic spreading in multilayer networks with simplicial complexes," *Chaos, Solitons Fractals*, vol. 164, Nov. 2022, Art. no. 112734.
- [22] P. Huang, X.-L. Chen, M. Tang, and S.-M. Cai, "Coupled dynamic model of resource diffusion and epidemic spreading in time-varying multiplex networks," *Complexity*, vol. 2021, pp. 1–11, Mar. 2021.
- [23] S. C. Ferreira, C. Castellano, and R. Pastor-Satorras, "Epidemic thresholds of the susceptible-infected-susceptible model on networks: A comparison of numerical and theoretical results," *Phys. Rev. E, Stat. Phys. Plasmas Fluids Relat. Interdiscip. Top.*, vol. 86, no. 4, Oct. 2012, Art. no. 041125.
- [24] M. Boguñá, C. Castellano, and R. Pastor-Satorras, "Langevin approach for the dynamics of the contact process on annealed scale-free networks," *Phys. Rev. E, Stat. Phys. Plasmas Fluids Relat. Interdiscip. Top.*, vol. 79, no. 3, Mar. 2009, Art. no. 036110.
- [25] S. Gómez, A. Arenas, J. Borge-Holthoefer, S. Meloni, and Y. Moreno, "Discrete-time Markov chain approach to contact-based disease spreading in complex networks," *EPL, Europhys. Lett.*, vol. 89, no. 3, p. 38009, Feb. 2010.

- [26] P. Yang, Z. Xu, J. Feng, and X. Fu, "Feedback pinning control of collective behaviors aroused by epidemic spread on complex networks," *Chaos*, vol. 29, no. 3, Mar. 2019, Art. no. 033122.
- [27] M. Liu, R. Zhang, and B. Xie, "The impact of behavioral change on the epidemic under the benefit comparison," *Math. Biosci. Eng.*, vol. 17, no. 4, pp. 3412–3425, 2020.
- [28] R. Zhang, M. Liu, and B. Xie, "The analysis of discrete-time epidemic model on networks with protective measures on game theory," *Chaos, Solitons Fractals*, vol. 158, May 2022, Art. no. 112029.
- [29] M. Robinson and N. I. Stilianakis, "A model for the emergence of drug resistance in the presence of asymptomatic infections," *Math. Biosci.*, vol. 243, no. 2, pp. 163–177, Jun. 2013.
- [30] S. Ottaviano, M. Sensi, and S. Sottile, "Global stability of SAIRS epidemic models," *Nonlinear Anal., Real World Appl.*, vol. 65, Jun. 2022, Art. no. 103501.
- [31] F. Z. Zhang, Matrix Theory: Basic Results and Techniques. New York, NY, USA: Springer, 2010, pp. 6–8.
- [32] S. Chen and Y. Ran, "Epidemic dynamics of two-pathogen spreading for pairwise models," *Mathematics*, vol. 10, 2022.
- [33] B. Yang, K.-K. Shang, M. Small, and N. Chao, "Information overload: How hot topics distract from news-COVID-19 spread in the U.S.," *Nat. Sci. Open*, vol. 2, no. 6, Jun. 2023, Art. no. 20220051.



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