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Research article

Markov modeling and performance analysis of infectious diseases with asymptomatic patients

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Abstract: After over three years of COVID-19, it has become clear that infectious diseases are difficult to eradicate, and humans remain vulnerable under their influence in a long period. The presence of presymptomatic and asymptomatic patients is a significant obstacle to preventing and eliminating infectious diseases. However, the long-term transmission of infectious diseases involving asymptomatic patients still remains unclear. To address this issue, this paper develops a novel Markov process for infectious diseases with asymptomatic patients by means of a continuous-time level-dependent quasi-birth-and-death (QBD) process. The model accurately captures the transmission of infectious diseases by specifying several key parameters (or factors). To analyze the role of asymptomatic and symptomatic patients in the infectious disease transmission process, a simple sufficient condition for the stability of the Markov process of infectious diseases is derived using the mean drift technique. Then, the stationary probability vector of the QBD process is obtained by using RG-factorizations. A method of using the stationary probability vector is provided to obtain important performance measures of the model. Finally, some numerical experiments are presented to demonstrate the model's feasibility through analyzing COVID-19 as an example. The impact of key parameters on the system performance evaluation and the infectious disease transmission process are analyzed. The methodology and results of this paper can provide theoretical and technical support for the scientific control of the long-term transmission of infectious diseases, and we believe that they can serve as a foundation for developing more general models of infectious disease transmission.

Keywords: coronavirus disease 2019 (COVID-19); asymptomatic patients; Markov process; quasi-birth-and-death process (QBD); performance evaluation

1. Introduction

In recent years, large-scale epidemic outbreaks involving asymptomatic patients have attracted widespread attention, especially COVID-19 since the end of 2019, which poses a huge threat to global human health, life safety and economic development. Due to the significant hazards caused by infectious diseases, countries have to adopt appropriate prevention and control strategies to contain virus transmission once an outbreak occurs. For example, some countries have implemented measures such as closing schools and businesses to restrict gatherings, and locking down cities to limit mobility (UK Government. Coronavirus (COVID-19): guidance and support. https://www.gov.uk/coronavirus. Accessed March 17, 2023). Evidently, rapid control of the outbreak became an important issue.

Asymptomatic carriers of infectious diseases pose significant challenges for epidemic control, especially the COVID-19 pandemic. They are individuals who tested positive for the disease but do not have any clinical symptoms that they can perceive [1]. Based on this meaning of asymptomatic patients, we refer to pre-symptomatic patients and those who are asymptomatic throughout the duration of their illness collectively as asymptomatic patients in this work. While asymptomatic carriers may not be screened or diagnosed, they can still carry the virus and become a potential source of infection. Those patients may have longer exposure times and contact with more people than symptomatic patients, as they are unaware of their infection. Therefore, asymptomatic carriers are difficult to detect and isolate in a timely manner so that they directly cause the high transmission rate due to their hidden transmission characteristics. Neglecting the infection caused by asymptomatic carriers has led to continued transmission and repeated outbreaks in some countries and regions [2]. Some studies have shown that the existence of asymptomatic carriers is one of the important and key reasons for the difficulty in controlling the COVID-19 pandemic [3–5]. Therefore, it is an important topic to study the mechanisms of transmission with asymptomatic carriers and to find the key factors during the long-term transmission process of infectious diseases, so as to predict epidemic trends and develop effective prevention and control strategies.

At present, research on the mechanisms of transmission with asymptomatic carriers mainly focuses on two classes: qualitative analysis and quantitative analysis. We review recent studies on infectious diseases with asymptomatic patients through taking COVID-19 as an example. For the first class, several scholars reviewed the characteristics of asymptomatic COVID-19 patients, mainly concerning contagiousness [6–8], infectious characteristics [8–10] and proportion of asymptomatic patients among all COVID-19 patients [8]. In terms of contagiousness, Nelson [11] and Yu et al. [12] found that asymptomatic patients are also infectious and there are cases of interpersonal infection. For the infectious characteristics study, Han et al. [13] and Hu et al. [14] found that asymptomatic patients had longer durations of virus presence, which posed a greater risk of infection. He et al. [15] found that asymptomatic patients had lower infection rates than symptomatic patients, and that patients infected by asymptomatic patients were more likely to be asymptomatic patients. Chen Yi et al. [16] found that the viral infection rates were about 6.30% and 4.11% for contact with symptomatic and asymptomatic patients, respectively. Zhou et al. [17] found that some asymptomatic patients transformed into symptomatic patients, and some patients remained asymptomatic patients. Kimball et al. [18] studied the proportion of asymptomatic patients who are transformed to symptomatic patients. Lauer et al. [19] observed that asymptomatic patients transformed to symptomatic patients after approximately 5 days of latency. For the problem of the proportion of asymptomatic patients, several authors investigated the proportion of asymptomatic patients in various sizes of the population of COVID-19 patients. Wu et al. [20] reviewed 72,314 patients with COVID-19 and found that the proportion of asymptomatic patients was about 1%. Using an example of 565 COVID-19 patients,

Nishiura et al. [21] employed a Bayes theorem to estimate that the proportion of asymptomatic patients was 30.8%. Day [22] illustrated that the proportion of asymptomatic COVID-19 patients may reach four-fifths based on a published sample of 166 COVID-19 patients in China.

For the second class of study, the quantitative study, considering the transmission process of COVID-19 with asymptomatic patients, most research used dynamical system theory, simulation or the complex network approach. Using dynamical systems theory, Sang Maosheng et al. [23] and Agrawal et al. [24] developed a modified susceptible–infectious–recovered (SIR) model to fit the transmission trend of COVID-19 considering asymptomatic patients. Masaki et al. [25] constructed a new mathematical model called SIIR model and found that the conditions for population immunity became more restrictive when considering the limited duration of immunity in asymptomatic patients. Some scholars [26–30] explored the effect of infection rates in asymptomatic patients on the transmission of COVID-19 or the effectiveness of control strategies by the basic reproduction number R_0. Riyapan et al. [31] and Iaa et al. [32] proposed a modified SIR model considering asymptomatic patients and found that COVID-19 will die out when the basic reproduction number is less than 1 and will continue to exist when the basic reproduction number is bigger than 1. Simulation methods have also been used to study the COVID-19 transmission process, supporting the inference of potentially infected individuals based on the temporal and location information of their activities. Zhan et al. [33] estimated the basic reproduction number for the COVID-19 transmission process on the Diamond Princess by the simulation method. Yu et al. [34] estimated the number of asymptomatic patients and their transmission rate based on a machine learning simulation method. By means of the complex network approach, some scholars studied the COVID-19 transmission process. Chen et al. [35] set up a novel COVID-19 transmission model through considering asymptomatic patients based on the complex network approach to make accurate forecasts of epidemic trends in the presence of incomplete information. Stella et al. [36] developed a complex network model to study the impact of asymptomatic patients and control measures on transmission while capturing the interactions of symptomatic and asymptomatic COVID-19 patients separately.

To study the transmission process of COVID-19 with asymptomatic patients, the construction of SIR models by using dynamic systems theory is necessary to parameterize the probability of exposure, infection, recovery, death, etc. for representing the change in the health status of the infected person. It is inferior for individual spatial movement and interaction processes, i.e., the heterogeneity of contact exposure among individuals. Therefore, the SIR-based models are only suitable for studying the infectious disease transmission process in a short time [37,38]. The simulation method is closer to the real situation and can support the analysis of temporal transmission mechanisms of infectious diseases and the design of appropriate interventions. However, the acquisition of a large amount of real individual trajectory data as a support is a challenge [39]. Using the complex network to simulate the contact among transmission subjects can reflect the variability of individual contacts in the social network structure. Unfortunately, it cannot represent the spatio-temporal spread of the disease well [38]. To quantitatively study the long-term transmission of infectious diseases with asymptomatic patients, it is necessary to develop a new mathematical model that depicts the characteristics of people with cross-regional movement in real life, that is, an infectious disease transmission process with spatial movement and interaction, and there is no limit to the number of susceptible persons. To our knowledge, there is still a lack of effective methods to discuss the stability of the long-term transmission and conduct system, and to provide performance analysis for the infectious disease transmission process with asymptomatic patients.

The motivation of this work is to study a long-term transmission process with realistic characteristics that can be used to analyze the mechanism of asymptomatic carriers. To this end, we

develop a new two-dimensional Markov process of infectious disease by means of the level-dependent continuous-time quasi-birth-and-death (QBD) process with infinite-state space. This Markov process can characterize interindividual interactions on infinite scales for patient groups containing both symptomatic and asymptomatic patients [40,41]. On the one hand, this Markov process, as a stochastic process, can better reflect stochasticity and suitability for stability analysis compared to deterministic models. On the other hand, the two-dimensional structure can reflect the complex interindividual interactions among symptomatic and asymptomatic patients during spatio-temporal transmission. Therefore, it is suitable for analyzing the long-term transmission process of infectious diseases with asymptomatic patients. Based on the actual situation of infectious diseases and some studies in the literature, the level on the infinite state space is used to represent symptomatic patients and the phase on the finite state space is used to represent asymptomatic patients. Such a level-dependent Markov process can reflect the different stages of the long-term transmission process of infectious diseases. Furthermore, to deal with the level-dependent QBD processes of infectious disease with infinite-state space, we use the RG-factorizations to find a feasible solution. Additionally, we develop two effective RG-factorization algorithms to obtain the feasible solution that can numerically analyze performance measures of this system of infectious disease.

The main contributions of this study are summarized as follows:

1) We develop a new two-dimensional continuous-time QBD process with an infinite-state space in the context that infectious diseases spread over a long time. This Markov process represents the status and transfer rate in different types of patients in the long-term and also expresses the spatial movement and interaction of individuals.

2) We provide a method based on the mean drift technique to obtain a simple sufficient condition for the stability of the proposed Markov process. The stationary probability vector is obtained by using RG-factorization. Additionally, we develop two algorithms to obtain and discuss some key performance measures of this system.

3) We conduct some numerical analysis to verify the feasibility of the proposed model by using the real data in the context of the COVID-19 epidemic and discuss how some key parameters affect the key performance measures of this system.

The remainder of this paper is organized as follows. In Section 2, we describe the infectious disease transmission process with asymptomatic patients and introduce the basic parameters. Section 3 establishes a Markov process of infectious disease considering asymptomatic patients. Section 4 obtains the stationary probability vector of the proposed model by using RG-factorizations and computes some key performance measures of the system by means of the stationary probability vector. Section 5 designs two algorithms to get the stationary probability vector and analyzes performance measures of the system. Section 6 provides numerical cases to verify the feasibility of the model and analyzes the impact of some key parameters on performance measures and infectious disease transmission. Section 7 makes conclusions and presents future research.

2. Problem description

In this section, we describe the infectious disease transmission process with asymptomatic patients, and give the relevant symbols and parameters used in the modeling.

In the infectious disease transmission process with asymptomatic patients, there are four types of patients: symptomatic, asymptomatic, cured and dead patients as shown in Figure 1. Symptomatic patients refer to patients with confirmed infectious diseases who have relevant symptoms. According

to the meaning of asymptomatic patients, they include pre-symptomatic patients and patients who always had no relevant symptoms during their illness. Both symptomatic and asymptomatic patients are collectively referred to as infected patients. Asymptomatic patients may be transformed into symptomatic or cured patients. The end of disease progression in symptomatic and asymptomatic patients may be cured or death.

Figure 1. Four types of patients in the infectious disease transmission process with asymptomatic patients. The icon defined by the patient type in this figure is represented consistently throughout the next figures.

In the next, we give the detailed transmission process and assumptions, which are used to set up a Markov process of infectious disease.

1) **The first appearance process of patients:** We assume that the interval length of the number of symptomatic patients from 0 to 1 in an observation region is exponentially distributed with mean $1/\tau_A$, and the interval length of asymptomatic patients from 0 to 1 is exponentially distributed with mean $1/\tau_B$.

2) **The infection process of patients:** We assume that the infection process of patients is a Poisson process, which is consistent with Giri [42] and Alawiyah [43]. Both symptomatic and asymptomatic patients have the ability to transmit the disease to others, resulting in the generation of new cases that include both symptomatic and asymptomatic individuals. In the condition that a symptomatic case exists, the infection rates of generating a symptomatic and an asymptomatic patient are denoted by λ_{AA} and λ_{AB}, respectively. Similarly, in the condition that an asymptomatic case exists, the infection rates of generating a symptomatic and an asymptomatic patient are denoted by λ_{BA} and λ_{BB}, respectively. The infection process of an infectious disease is shown in Figure 2.

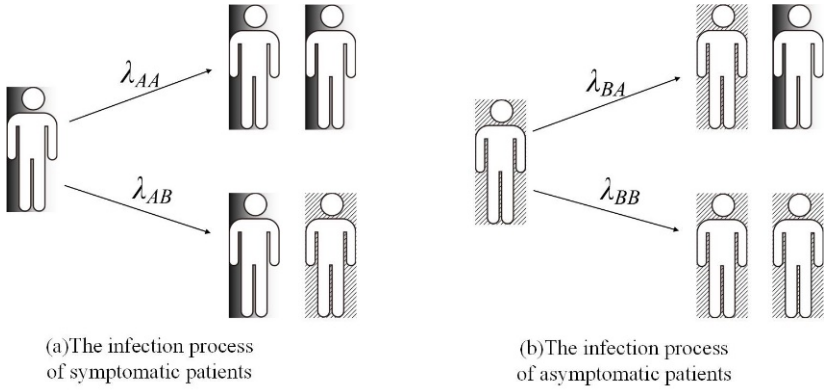

Figure 2. The infection process of a Markov process for an infectious disease. In Figure 2(a), the left side of the arrow indicates the situation before infection, the right side indicates the possible situation after infection, and the number on the arrow indicates the infection rate. Figure 2(b) is similar to Figure 2(a).

3) **The disappearance process of patients:** The findings of Davies [44,45] showed that the

infection period of COVID-19 fits an exponential distribution. In line with the literature, we assume that the interval length of symptomatic patients from being identified to being cured or death is exponentially distributed with mean $1/\mu_A$, and the interval length of asymptomatic patients from being identified to self-healing is exponentially distributed with mean $1/\mu_B$. The disappearance process of infected patients is shown in Figure 3. It is worth noting that the disappearance of symptomatic patients can be followed by cured patients or dead patients. However, the disappearance of asymptomatic patients can only be followed by cured patients. Realistically speaking, asymptomatic patients generally go through a symptomatic phase before they die.

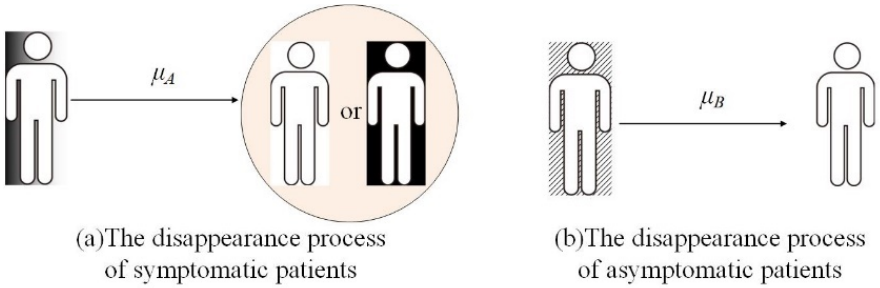

Figure 3. The disappearance process of infected patients. In Figure 3(a), the left side of the arrow indicates the situation before disappearance, the right side indicates the situation after disappearance, and the number on the arrow indicates the disappearance rate. Figure 3(b) is similar to Figure 3(a).

4) **The transformation process of patients:** Based on the setting that pre-symptomatic patients are also asymptomatic, some studies have found that asymptomatic patients can transform into symptomatic patients, but symptomatic patients cannot transform into asymptomatic patients [17,46,47]. Pasetto [48] shows that the mean duration that infected patients stay in the incubation period is exponentially distributed. Based on it, we assume that the interval length of transformation from asymptomatic patients to symptomatic patients is exponentially distributed with mean $1/\gamma$.

5) **Limited number of asymptomatic patients:** According to the literature [20–22], the asymptomatic patients gradually transform to symptomatic patients as the epidemic evolves. Therefore, we reasonably assume that the number of symptomatic and asymptomatic patients takes values in $\{0,1,2,\ldots\}$ and $\{0,1,2,\ldots,M\}$, respectively. Note that this assumption can simplify the model construction, proof and analysis.

6) **Independence:** We assume that all the random variables described above are independent of each other.

According to He et al. [15] and Chen Yi et al. [16], the infection rates and the interval length differ in different types of patients. Therefore, four different types of infection rates are assumed in Assumption (2), and the two different types of disappearing rates are assumed in Assumption (3). Although in this paper we describe the infectious disease transmission process by Poisson process and exponential distribution, our mathematical modeling can be easily extended, for example, from Poisson process to Markovian arrival processes (MAPS) in Assumption (2) and from exponential distribution to phase type (PH) distribution in Assumption (3).

Using the key parameters assumed above, in the next section we can develop a new Markov process of infectious disease with infinite-state space.

3. Markov process of infectious disease

In this section, we model the infectious disease transmission process as a Markov process. Specifically, this Markov process is a continuous-time level-dependent QBD process with an infinite state space.

Let $N_A(t)$ and $N_B(t)$ be the number of symptomatic patients and asymptomatic patients at time t, respectively. Based on the assumptions, $N_A(t) \in \{0,1,2,\ldots\}$ and $N_B(t) \in \{0,1,2,\ldots,M\}$ $\{(N_A(t), N_B(t)) : t \geq 0\}$. Therefore, it is known that $\{(N_A(t), N_B(t)) : t \geq 0\}$ is a continuous-time Markov process with a state space of

$$\Omega = \bigcup_{k=0}^{\infty} \text{Level } k, \tag{1}$$

where the Level k denotes the set of states in level k, for details:

$$\text{Level } k = \{(k,0),(k,1),(k,2),\ldots,(k,M)\}, k \geq 0. \tag{2}$$

Based on the problem description and using the states in all Levels, we can give the state transition relations for this Markov process $\{(N_A(t), N_B(t)) : t \geq 0\}$, as shown in Figure 4.

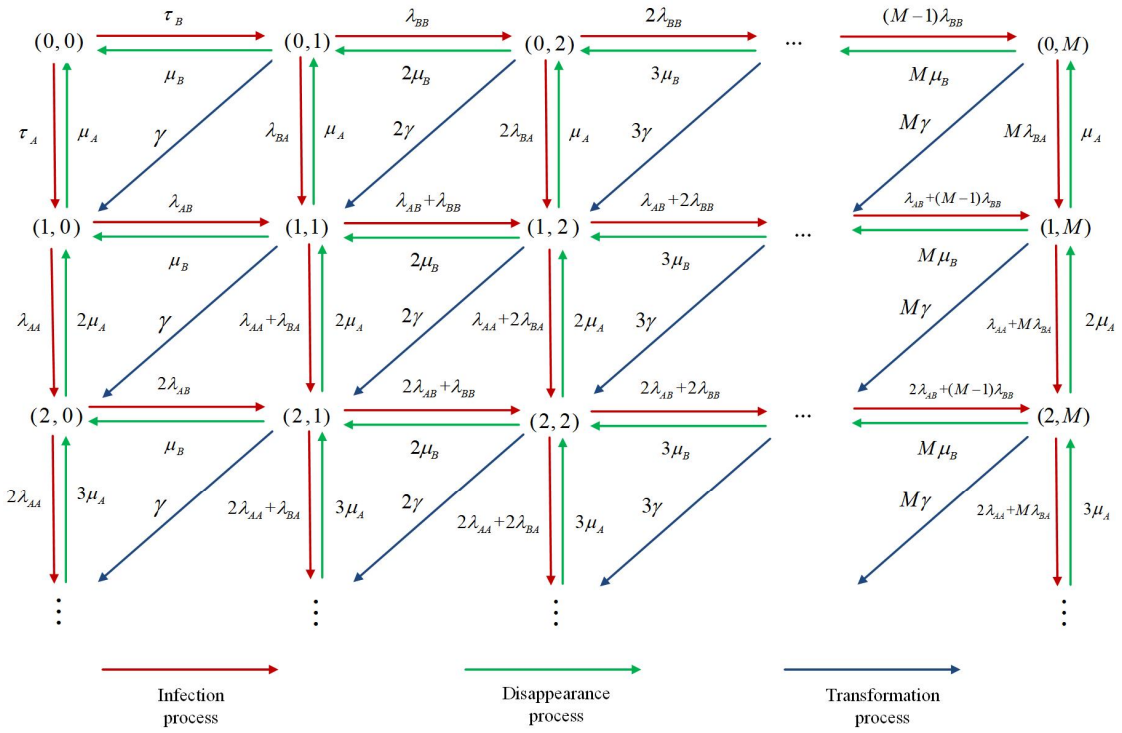

Figure 4. The state transition relations for the Markov process. In Figure 4, the infection process, the disappearance process and the transformation process are shown by red, green and blue arrows, respectively. If the system is in the state where the arrow starts at a certain moment, it will later transit to one of the states where the arrows end. The symbols on the arrows indicate the transition rates.

Further, according to the state transition relation given in Figure 4, the infinitesimal generator of this Markov process can be obtained. This infinitesimal generator contains a great deal of the information about our proposed Markov process, and the model analysis and solution in this paper are carried out based on this infinitesimal generator [49]. The infinitesimal generator can be written as

$$Q=\begin{pmatrix} A_{0,0} & A_{0,1} & & & & & \\ A_{1,0} & A_{1,1} & A_{1,2} & & & & \\ & A_{2,1} & A_{2,2} & A_{2,3} & & & \\ & & \ddots & \ddots & \ddots & & \\ & & & A_{n-1,n} & A_{n,n} & A_{n,n+1} & \\ & & & & \ddots & \ddots & \ddots \end{pmatrix}, \tag{3}$$

where $A_{i,j}$ are all square arrays of order $M+1$. To easily give the details of $A_{i,j}$, let $a_{n,i}=n(\mu_A+\lambda_{AA}+\lambda_{AB})+i(\gamma+\mu_B+\lambda_{BA}+\lambda_{BB})$, $b_{n,i}=n\lambda_{AB}+i\lambda_{BB}$ and $d_{n,i}=n\lambda_{AA}+i\lambda_{BA}$. Using $a_{n,i}$, $b_{n,i}$ and $d_{n,i}$, we can give the details of $A_{i,j}$ as follows:

$$A_{0,0}=\begin{pmatrix} -(\tau_A+\tau_B) & \tau_B & & & & \\ \mu_B & -a_{0,1} & b_{0,1} & & & \\ & 2\mu_B & -a_{0,2} & b_{0,2} & & \\ & & \ddots & \ddots & \ddots & \\ & & & (M-1)\mu_B & -a_{0,M-1} & b_{0,M-1} \\ & & & & M\mu_B & -a_{0,M} \end{pmatrix}, \tag{4}$$

$$A_{0,1}=\begin{pmatrix} \tau_A & & & & & \\ \gamma & d_{0,1} & & & & \\ & 2\gamma & d_{0,2} & & & \\ & & \ddots & \ddots & & \\ & & & (M-1)\gamma & d_{0,M-1} & \\ & & & & M\gamma & d_{0,M} \end{pmatrix}. \tag{5}$$

When $k\geq 1$,

$$A_{k,k-1}=\begin{pmatrix} k\mu_A & & & & & \\ & k\mu_A & & & & \\ & & k\mu_A & & & \\ & & & \ddots & & \\ & & & & k\mu_A & \\ & & & & & k\mu_A \end{pmatrix}, \tag{6}$$

$$A_{k,k}=\begin{pmatrix} -a_{k,0} & b_{k,0} & & & & \\ \mu_B & -a_{k,1} & b_{k,1} & & & \\ & 2\mu_B & -a_{k,2} & b_{k,2} & & \\ & & \ddots & \ddots & \ddots & \\ & & & (M-1)\mu_B & -a_{k,M-1} & b_{k,M-1} \\ & & & & M\mu_B & -a_{k,M} \end{pmatrix}, \tag{7}$$

$$A_{k,1}=\begin{pmatrix} d_{k,0} & & & & & \\ \gamma & d_{k,1} & & & & \\ & 2\gamma & d_{k,2} & & & \\ & & \ddots & \ddots & & \\ & & & (M-1)\gamma & d_{k,M-1} & \\ & & & & M\gamma & d_{k,M} \end{pmatrix}. \tag{8}$$

Based on this state transition relation and infinitesimal generator, we can know that the Markov process of infectious disease is a level-dependent QBD process with an infinite state space. Specifically, a state within a given level can only transition to other states in the same level or in the adjacent levels. Moreover, the process exhibits an infinite state space that arises from the infinite number of levels. Finally, the process is level-dependent, as the state transition matrices differ across levels.

For the level-dependent QBD process we propose, it is easy to prove its stability. When $\mu_A > \lambda_{AA}$, this QBD process of infectious disease (Q) is irreducible and positive recurrent. Thus, the QBD process of infectious disease is stable. We give details of the proof process in the appendix.

4. Stationary probability vector and performance measures

In this section, we present how to use the RG-factorization method to obtain the stationary probability vector of the QBD process proposed in this paper and provide some important performance measures of the model based on the stationary probability vector. RG-factorization is an efficient way of solving infinite dimensional (or large) systems of linear equations. More details about RG-factorization can be found in the literature [50,51].

Let $P_{i,j}(t)$ be the probability that the process is at state (i,j) at time t. We have

$$P_{i,j}(t)=P\{N_A(t)=i,N_B(t)=j\}. \tag{9}$$

When the QBD process of infectious disease is stable, let $\pi_{i,j}$ denote the stationary probability that the process is at state (i,j). $\pi_{i,j}$ can be obtained by

$$\pi_{i,j}=\lim_{t\to\infty}P_{i,j}(t), \tag{10}$$

After that, we denote the stationary probability vector that the process is at level i by $\boldsymbol{\pi}_i$. For any integer i = 0, 1, 2, …, we have

$$\boldsymbol{\pi}_i=\left(\pi_{i,0},\pi_{i,1},\pi_{i,2},\ldots,\pi_{i,M}\right), \tag{11}$$

and the stationary probability vector $\boldsymbol{\pi}$ of this process is

$$\boldsymbol{\pi} = (\boldsymbol{\pi}_0, \boldsymbol{\pi}_1, \boldsymbol{\pi}_2, \ldots). \tag{12}$$

Using the RG-factorizations method [49], we can calculate the stationary probability vector of this QBD process. Therefore, we give the $U-$, $R-$ and $G-$ measures of the QBD process respectively.

$$U_k = A_{k,k} + A_{k,k+1}(-U_{k+1}^{-1})A_{k+1,k}, k \geq 0, \tag{13}$$

$$R_k = A_{k,k+1}(-U_{k+1}^{-1}), k \geq 0, \tag{14}$$

$$G_k = (-U_{k+1}^{-1})A_{k,k-1}, k \geq 1. \tag{15}$$

Note that the matrix sequence $\{R_k, k \geq 0\}$ is the smallest non-negative solution to the system of nonlinear matrix Eq (16):

$$A_{k,k+1} + R_k A_{k+1,k+1} + R_k R_{k+1} A_{k+2,k+1} = 0, k \geq 0. \tag{16}$$

Similarly, the matrix sequence $\{G_k, k \geq 1\}$ is the smallest non-negative solution to the system of nonlinear matrix Eq (17):

$$A_{k,k+1} G_{k+1} G_k + A_{k,k} G_k + A_{k,k-1} = 0, k \geq 1. \tag{17}$$

Once the matrix sequence $\{R_k, k \geq 0\}$ or $\{G_k, k \geq 1\}$ is given, for $k \geq 0$ we have

$$\begin{aligned} U_k &= A_{k,k} + R_k A_{k+1,k} \\ &= A_{k,k} + A_{k,k+1} G_{k+1}. \end{aligned} \tag{18}$$

By following the method described in Chapter 1 of Li [49], we can factorize the infinitesimal generator of the level-dependent QBD process as

$$Q = (I - R_U) U_D (I - G_L), \tag{19}$$

where,

$$U_D = \text{diag}(U_o, U_1, U_2 \ldots), \tag{20}$$

$$R_U = \begin{pmatrix} 0 & R_0 & & \\ & 0 & R_1 & \\ & & 0 & \ddots \\ & & & \ddots \end{pmatrix}, \tag{21}$$

$$G_L = \begin{pmatrix} 0 & & & \\ G_1 & 0 & & \\ & G_2 & 0 & \\ & & \ddots & \ddots \end{pmatrix}. \tag{22}$$

The $\text{diag}(U_o, U_1, U_2 \ldots)$ denotes the diagonal matrix composed of U_o, U_1, U_2, ….

After that, using this UL-type RG-Factorization and R-measure sequence $\{R_k, k \geq 0\}$, we have

$$\tilde{\boldsymbol{\pi}}_k = \tilde{\boldsymbol{\pi}}_0 R_0 R_1 \cdots R_{k-1}, k \geq 1, \tag{23}$$

$$\boldsymbol{\pi}_k = c\tilde{\boldsymbol{\pi}}_k, k \geq 0, \tag{24}$$

where c is the normalization constant such that the sum of the stationary probabilities of all states is 1. And the positive constant c is uniquely given by

$$c = \frac{1}{\tilde{\boldsymbol{\pi}}_0 \mathbf{e} + \sum_{k=1}^{\infty} \tilde{\boldsymbol{\pi}}_0 R_0 R_1 \cdots R_{k-1} \mathbf{e}}. \tag{25}$$

In Eq (23), $\tilde{\boldsymbol{\pi}}_k$ represents the stationary probabilities vector of level k before normalization. $\tilde{\boldsymbol{\pi}}_0$ can be uniquely determined by the system of linear Eq (40).

$$\begin{cases} \tilde{\boldsymbol{\pi}}_0 (A_{0,0} + R_0 A_{1,0}) = 0 \\ \tilde{\boldsymbol{\pi}}_0 \mathbf{e} = 1 \end{cases}. \tag{26}$$

In the remainder of this section, we provide performance analysis of the infectious disease transmission process with asymptomatic patients by means of the stationary probability vector of the QBD process of infectious diseases. Therefore, we can use this stationary probability vector $\boldsymbol{\pi}$ to provide some useful performance measures as follows:

i. Average number of symptomatic patients

$$\begin{aligned} E[N_A] &= \sum_{k=0}^{\infty} k\boldsymbol{\pi}_k \mathbf{e} \\ &= \sum_{k=1}^{\infty} kc\tilde{\boldsymbol{\pi}}_0 R_0 R_1 \cdots R_{k-1} \mathbf{e}, \end{aligned} \tag{27}$$

where, $N_A = \lim_{t \to \infty} N_A(t)$, a.s.

ii. Average number of asymptomatic patients

$$\begin{aligned} E[N_B] &= \sum_{k=0}^{\infty} \boldsymbol{\pi}_k \mathbf{f}_B \\ &= c\tilde{\boldsymbol{\pi}}_0 \mathbf{f}_B + \sum_{k=1}^{\infty} c\tilde{\boldsymbol{\pi}}_0 R_0 R_1 \cdots R_{k-1} \mathbf{f}_B, \end{aligned} \tag{28}$$

where, $N_B = \lim_{t \to \infty} N_B(t)$, a.s.; $\mathbf{f}_B = (0,1,2,\ldots,M)^T$.

iii. Average infection rate from symptomatic patients to symptomatic patients

$$\begin{aligned} r_{A\to A} &= \lambda_{AA}(\pi_{1,0}+2\pi_{2,0}+3\pi_{3,0}+\cdots+\pi_{1,1}+2\pi_{2,1}+3\pi_{3,1}+\cdots+ \\ &\quad \pi_{1,2}+2\pi_{2,2}+3\pi_{3,2}+\cdots+\pi_{1,M}+2\pi_{2,M}+3\pi_{3,M}+\cdots) \\ &= \lambda_{AA}(\boldsymbol{\pi}_1\mathbf{e}+2\boldsymbol{\pi}_2\mathbf{e}+3\boldsymbol{\pi}_3\mathbf{e}+\cdots) \\ &= \lambda_{AA}\sum_{k=1}^{\infty}k\boldsymbol{\pi}_k \\ &= \lambda_{AA}E[N_A]. \end{aligned} \tag{29}$$

iv. Average infection rate from symptomatic patients to asymptomatic patients

$$\begin{aligned} r_{A\to B} &= \lambda_{AB}(\pi_{1,0}+2\pi_{2,0}+3\pi_{3,0}+\cdots+\pi_{1,1}+2\pi_{2,1}+3\pi_{3,1}+\cdots+ \\ &\quad \pi_{1,2}+2\pi_{2,2}+3\pi_{3,2}+\cdots+\pi_{1,M}+2\pi_{2,M}+3\pi_{3,M}+\cdots) \\ &= \lambda_{AB}(\boldsymbol{\pi}_1\mathbf{e}+2\boldsymbol{\pi}_2\mathbf{e}+3\boldsymbol{\pi}_3\mathbf{e}+\cdots) \\ &= \lambda_{AB}\sum_{k=1}^{\infty}k\boldsymbol{\pi}_k \\ &= \lambda_{AB}E[N_A]. \end{aligned} \tag{30}$$

v. Average infection rate from asymptomatic patients to symptomatic patients

$$\begin{aligned} r_{B\to A} &= \lambda_{BA}(\pi_{0,1}+\pi_{1,1}+\pi_{2,1}+\cdots+2\pi_{0,2}+2\pi_{1,2}+2\pi_{2,2}+\cdots+ \\ &\quad 3\pi_{0,3}+3\pi_{1,3}+3\pi_{2,3}+\cdots+M\pi_{0,M}+M\pi_{1,M}+M\pi_{2,M}+\cdots) \\ &= \lambda_{BA}(\boldsymbol{\pi}_0\mathbf{f}_B+\boldsymbol{\pi}_1\mathbf{f}_B+\boldsymbol{\pi}_2\mathbf{f}_B+\cdots) \\ &= \lambda_{BA}\sum_{k=0}^{\infty}\boldsymbol{\pi}_k\mathbf{f}_B \\ &= \lambda_{BA}E[N_B]. \end{aligned} \tag{31}$$

vi. Average infection rate from asymptomatic patients to asymptomatic patients

$$\begin{aligned} r_{B\to B} &= \lambda_{BB}(\pi_{0,1}+\pi_{1,1}+\pi_{2,1}+\cdots+2\pi_{0,2}+2\pi_{1,2}+2\pi_{2,2}+\cdots+ \\ &\quad 3\pi_{0,3}+3\pi_{1,3}+3\pi_{2,3}+\cdots+(M-1)\pi_{0,M}+(M-1)\pi_{1,M}+(M-1)\pi_{2,M}+\cdots) \\ &= \lambda_{BB}(\boldsymbol{\pi}_0\mathbf{g}_B+\boldsymbol{\pi}_1\mathbf{g}_B+\boldsymbol{\pi}_2\mathbf{g}_B+\cdots) \\ &= \lambda_{BB}\sum_{k=0}^{\infty}\boldsymbol{\pi}_k\mathbf{g}_B, \end{aligned} \tag{32}$$

where, $\mathbf{g}_B=(0,1,2,\ldots,M-1,0)^T$.

vii. Average transition rate from asymptomatic patients to symptomatic patients

$$\begin{aligned} r_{BA} &= \gamma(\pi_{0,1}+\pi_{1,1}+\pi_{2,1}+\cdots+2\pi_{0,2}+2\pi_{1,2}+2\pi_{2,2}+\cdots+ \\ &\quad 3\pi_{0,3}+3\pi_{1,3}+3\pi_{2,3}+\cdots+M\pi_{0,M}+M\pi_{1,M}+M\pi_{2,M}+\cdots) \\ &= \gamma(\boldsymbol{\pi}_0\mathbf{f}_B+\boldsymbol{\pi}_1\mathbf{f}_B+\boldsymbol{\pi}_2\mathbf{f}_B+\cdots) \\ &= \gamma\sum_{k=0}^{\infty}\boldsymbol{\pi}_k\mathbf{f}_B \\ &= \gamma E[N_B]. \end{aligned} \tag{33}$$

5. Design of solution algorithms

This section provides two efficient algorithms for computing the stationary probability vector of the QBD process of infectious disease. And we use this stationary probability vector to compute the important performance measures of the QBD process.

Given the infinite state space of the QBD process, it is critical to first identify the maximum truncation level and the *R*-measure sequence $\{R_k, k \geq 0\}$ in order to obtain the stationary probability vector ($\boldsymbol{\pi}$). For this purpose, we propose an algorithm based on the method of Bright and Taylor [52], which enables the determination of the maximum truncation level K and all *R*-measure $\{R_k, k \geq 0\}$.

Based on Bright and Taylor [52], we derive two key results as follows:

a) An important UD-type representation of R_k.

$$R_k = \sum_{l=0}^{\infty} U_k^l \prod_{i=0}^{l-1} D_{k+2^{l-i}}^{l-1-i}, k \geq 1, \tag{34}$$

where

$$U_k^0 = A_{k,k+1}(-A_{k+1,k+1})^{-1}, k \geq 1, \tag{35}$$

$$D_k^0 = A_{k-1,k}(-A_{k-1,k-1})^{-1}, k \geq 1, \tag{36}$$

$$U_k^{l+1} = U_k^l U_{k+2^l}^l [I - U_{k+2^{l+1}}^l D_{k+3\cdot 2^l}^l - D_{k+2^{l+1}}^l U_{k+2^l}^l]^{-1}, k \geq 0, \tag{37}$$

$$D_k^{l+1} = D_k^l D_{k-2^l}^l [I - U_{k-2^{l+1}}^l D_{k-2^l}^l - D_{k-2^{l+1}}^l U_{k-3\cdot 2^l}^l]^{-1}, k \geq 0. \tag{38}$$

Note that, using this UD-type representation, the rate matrix $\{R_k, k \geq 1\}$ is determined directly from the original data matrices $A_{k,k+1}$, $A_{k+1,k+1}$, $A_{k-1,k}$, and $A_{k-1,k-1}$, $k \geq 1$.

b) A backward iterative recursive representation of R_k.

As soon as R_K is obtained from the UD-type representation, we have

$$R_k = A_{k,k+1}(-A_{k+1,k+1} - R_{k+1}A_{k+1,k+2})^{-1}, k = K-1, K-2, \ldots, 1, 0. \tag{39}$$

Based on a) and b), we propose an algorithm to determine the appropriate maximum truncation level K and obtain the *R*-measure $\{R_k, 0 \leq k \leq K\}$.

To determine the maximum truncation level K, we iteratively compute the sum of the stationary probabilities of the level K until it is sufficiently small, i.e., $\pi_K \mathbf{e} < \varepsilon$. ε denotes the controllable accuracy by the user, where we make $\varepsilon = 10^{-20}$. In addition, we define a sequence of positive integers $\{\varsigma_k : k \geq 0\}$ for iterative computation. The algorithm for determining the maximum truncation level and the *R*-measure is summarized in Algorithm 1.

Algorithm 1: Calculate K and R_k

Input: Parameter λ_{AA}, λ_{AB}, λ_{BA}, λ_{BB}, γ, μ_A and μ_B.

Output: K and R_k

1) **Step 0:** Initialization

Let $\varsigma_n = 10n$ and initialize setting n =1 as the initial value.

2) **Step 1:** Calculate the rate matrix R_K

Let $K = \varsigma_n$, and calculate R_K according to Eq (34).

3) **Step 2:** Determine other rate matrices ($\{R_k, 0 \le k \le K-1\}$) by backward iteration

Compute of R_{K-1}, R_{K-2}, …, R_2, R_1, R_0 according to Eq (39).

4) **Step 3:** Compute the stationary probability vector $\tilde{\boldsymbol{\pi}}_0$

Compute $\tilde{\boldsymbol{\pi}}_0$ by solving the linear equation system (26)

5) **Step 4:** Calculate the normalization constant c

Using the vector $\tilde{\boldsymbol{\pi}}_0$ and the R-measure $\{R_l, 0 \le l \le K\}$, we can compute

$$c = \frac{1}{\tilde{\boldsymbol{\pi}}_0 \mathbf{e} + \sum_{k=1}^{K+1} \tilde{\boldsymbol{\pi}}_0 R_0 R_1 \cdots R_{k-1} \mathbf{e}}. \tag{40}$$

6) **Step 5:** Check if the accuracy is satisfied

If for a positive integer $K = \varsigma_n$, it satisfies

$$\boldsymbol{\pi}_{K+1} \mathbf{e} < \varepsilon, \tag{41}$$

(called stop condition), where $\boldsymbol{\pi}_{K+1} = c\tilde{\boldsymbol{\pi}}_0 R_o R_1 \cdots R_K$, then let $K = \varsigma_n$, and go to Step 6. Otherwise, let $n = n+1$ and substitute to calculate $\varsigma_n = 10(n+1)$, return to Step 1;

7) **Step 6:** Output K and $\{R_k, 0 \le k \le K\}$

The algorithm stops, and a maximum truncation level $K = \varsigma_n$ and a sequence of R-measures $\{R_k, 0 \le k \le K\}$ are obtained as its output.

Once we have determined the maximum truncation level K and R-measure $\{R_k, 0 \le k \le K\}$ using Algorithm 1, we need to further compute the stationary probability vector and some other important performance measures of the model. Therefore, we design Algorithm 2 as follows.

Algorithm 2: Compute stationary probability vector $\boldsymbol{\pi}$ and performance measures

Input: Parameter K, $\{R_l, 0 \le l \le K\}$, $\tilde{\boldsymbol{\pi}}_0$, and c.

Output: $E[N_A]$, $E[N_B]$, $r_{A\to A}$, $r_{A\to B}$, $r_{B\to A}$, $r_{B\to B}$ and r_{BA}

1) **Step 1:** Calculate the stationary probability vector

When $1 \le k \le K+1$, based on Eqs (23) and (24), we can compute

$$\boldsymbol{\pi}_k = c\tilde{\boldsymbol{\pi}}_0 R_0 R_1 \cdots R_{k-1}, \tag{42}$$

and according to Eq (26), we can calculate

$$\boldsymbol{\pi}_0 = c\tilde{\boldsymbol{\pi}}_0. \tag{43}$$

Continued on next page

Algorithm 2: Compute stationary probability vector $\boldsymbol{\pi}$ and performance measures

2) **Step 2:** Calculate the important performance measures

We use Eqs (44)–(50) in Algorithm 2 to calculate the measures $E[N_A]$, $E[N_B]$, $r_{A\to A}$, $r_{A\to B}$, $r_{B\to A}$, $r_{B\to B}$, r_{BA}.

$$E[N_A]=\sum_{l=0}^{K} l\boldsymbol{\pi}_l\mathbf{e}, \tag{44}$$

$$E[N_B]=\sum_{l=0}^{K} \boldsymbol{\pi}_l\mathbf{f}_B, \tag{45}$$

$$r_{A\to A}=E[N_A]\cdot\lambda_{AA}, \tag{46}$$

$$r_{A\to B}=E[N_A]\cdot\lambda_{AB}, \tag{47}$$

$$r_{B\to A}=E[N_B]\cdot\lambda_{BA}, \tag{48}$$

$$r_{B\to B}=\sum_{k=0}^{K} \boldsymbol{\pi}_k\mathbf{g}_B\cdot\lambda_{BB}, \tag{49}$$

$$r_{BA}=E[N_B]\cdot\gamma. \tag{50}$$

3) **Step 3:** Output the important performance measures

Stop algorithm, output these performance measures: $E[N_A]$, $E[N_B]$, $r_{A\to A}$, $r_{A\to B}$, $r_{B\to A}$, $r_{B\to B}$, r_{BA}.

6. Numerical experiments

In this section, we give some numerical examples to demonstrate the feasibility of the proposed model in this paper and highlight the impact of key parameters on some important system performance measures.

6.1. Data setting

We conduct numerical experiments using COVID-19 as an example to verify the feasibility of the model. As obtaining actual data about asymptomatic patients can be challenging from publicly available information, we set the benchmark values of the model parameters based on previous studies. Specifically, He et al. [15] reported that the infection risk ratio between symptomatic and asymptomatic patients is approximately 3.9:1, i.e., $(\lambda_{AA}+\lambda_{AB})/(\lambda_{BA}+\lambda_{BB})=3.9/1$. In addition, about 85% of patients infected by symptomatic patients are symptomatic themselves, leading to $\lambda_{AA}/\lambda_{AB}=0.85/0.15$. Furthermore, roughly 50% of patients infected by asymptomatic patients become symptomatic, i.e., $\lambda_{BA}/\lambda_{BB}=0.5/0.5$. According to the results of Lauer et al. [18], the average duration for asymptomatic patients to become symptomatic is 5 days, which we make the parameter $\gamma=1/5$. We assume that these values remain constant throughout the observation period, which corresponds to the homogeneous Markov process.

After consulting relevant experts, referencing the research findings from literature [15,18], and taking into account the system stability conditions, we set the parameter values to be as follows: $\tau_A=1.5$, $\tau_B=3$, $\lambda_{AA}=3.4$, $\lambda_{AB}=0.6$, $\lambda_{BB}=0.51$, $\lambda_{BA}=0.51$, $\gamma=0.2$, $\mu_A=6$ and $\mu_B=1$. Subsequently, we give some numerical experiments to investigate the impact of various parameters on the average number of symptomatic and asymptomatic patients. We also analyze the effect of non-drug

interventions and vaccines. Note that, the parameters $r_{A\to A}$, $r_{A\to B}$, $r_{B\to A}$, $r_{B\to B}$ and r_{BA} are closely related to $E[N_A]$ and $E[N_B]$ according to Eqs (29)–(33), thus we only observe $E[N_A]$ and $E[N_B]$ in our numerical experiments.

6.2. Experiment results

1) Case 1: Let's take λ_{BB} as 0.3, 0.5 and 0.7, respectively, and observe the impact of λ_{AA} varying in the interval [2.9, 3.9] on $E[N_A]$ and $E[N_B]$. The numerical results are shown in Figure 5.

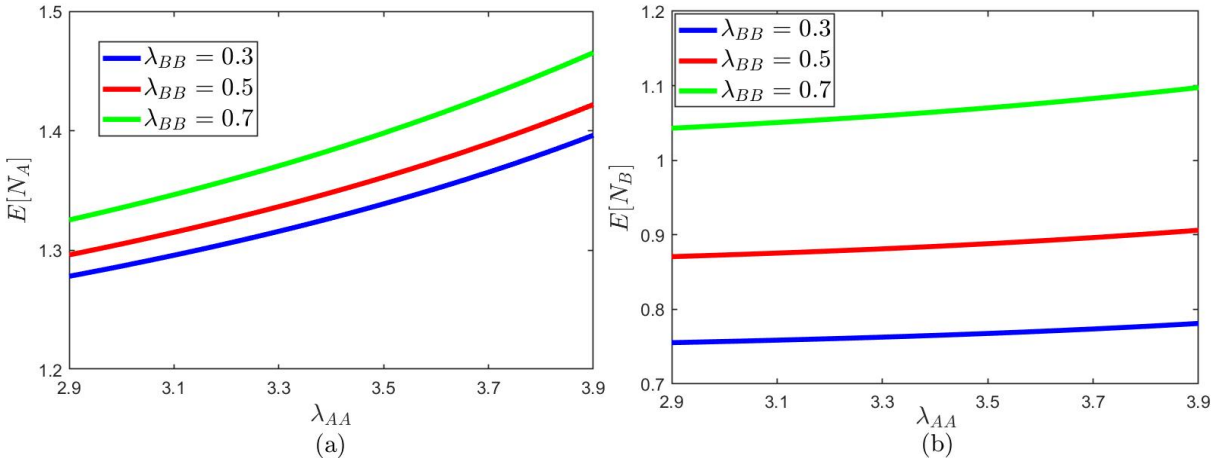

Figure 5. The impact of λ_{AA} on the average number of COVID-19 patients.

Figure 5 shows the impact of the infection rate of symptomatic patients infecting and generating symptomatic patients (λ_{AA}) on the average number of symptomatic patients and asymptomatic patients when the system is stable. The blue, red and green lines in Figure 5 represent the cases in which the rate of asymptomatic patients infecting and generating asymptomatic patients (λ_{BB}) is set as 0.3, 0.5 and 0.7, respectively. In Figure 5(a), each line shows that the average number of symptomatic patients increases as λ_{AA} increases. In addition, by comparing the three lines, we can observe that a larger λ_{BB} leads to a higher average number of symptomatic patients. Similarly, Figure 5(b) shows that the average number of asymptomatic patients also increases as λ_{AA} and λ_{BB} increase.

Based on the results shown in Figure 5, it can be concluded that the proposed method is feasible for modeling the long-term infectious disease transmission process. We can estimate the average number of symptomatic and asymptomatic patients by using our proposed model. The numerical experiments demonstrate that decreasing the infection rate of both symptomatic and asymptomatic patients can decrease the average number of symptomatic and asymptomatic patients. This suggests that certain non-drug interventions, such as implementing social distancing measures, can effectively control the transmission of infectious diseases by reducing the infection rates.

2) Case 2: Let's take μ_B as 1.1, 1.3 and 1.5, respectively, and observe the impact of μ_A varying in the interval [5, 6] on $E[N_A]$ and $E[N_B]$. The numerical results are shown in Figure 6.

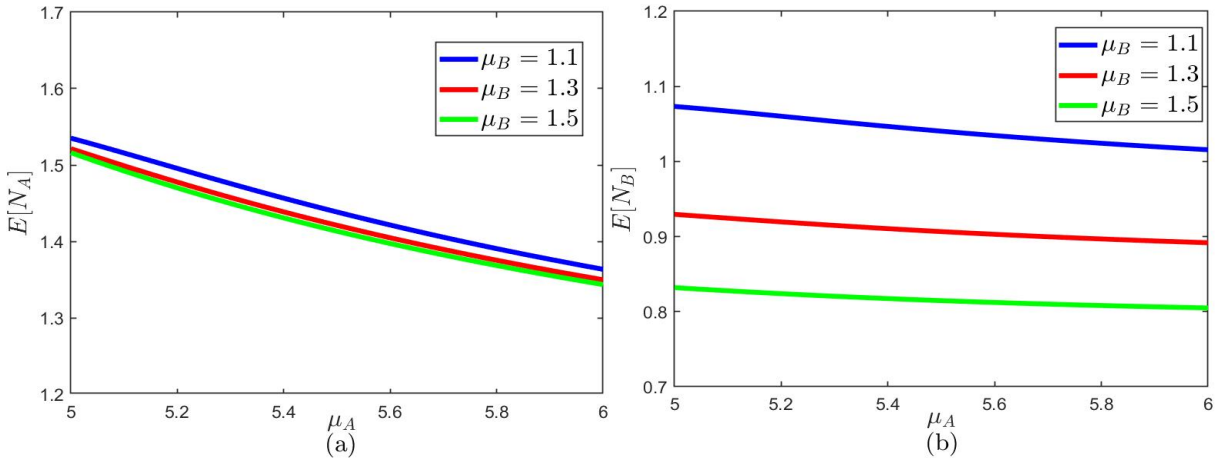

Figure 6. The effect of μ_A on the average number of COVID-19 patients.

Figure 6 shows the impact of the disappearance rate of symptomatic patients (μ_A) on the average number of symptomatic patients and asymptomatic patients. The blue, red and green lines in Figure 6 represent the cases in which the disappearance rate of asymptomatic patients (μ_B) is set as 1.1, 1.3 and 1.5, respectively. Each line in Figure 6(a) shows that the average number of symptomatic patients decreases as μ_A increase. In addition, comparing the three lines shows that the larger the value of μ_B, the smaller the average number of symptomatic patients. Similarly, Figure 6(b) shows that the average number of asymptomatic patients also decreases as μ_A and μ_B increase.

Based on the results in Figure 6, increasing the disappearance rate of symptomatic and asymptomatic patients can result in a decrease in the average number of symptomatic and asymptomatic patients. This shows the effectiveness of non-drug interventions, such as screening and isolation, in controlling the spread of infectious diseases by increasing the disappearance rates.

3) Case 3: Let's take μ_B as 1.1, 1.3 and 1.5, respectively, and observe the impact of γ varying in the interval [0, 1] on $E[N_A]$ and $E[N_B]$. The numerical results are shown in Figure 7.

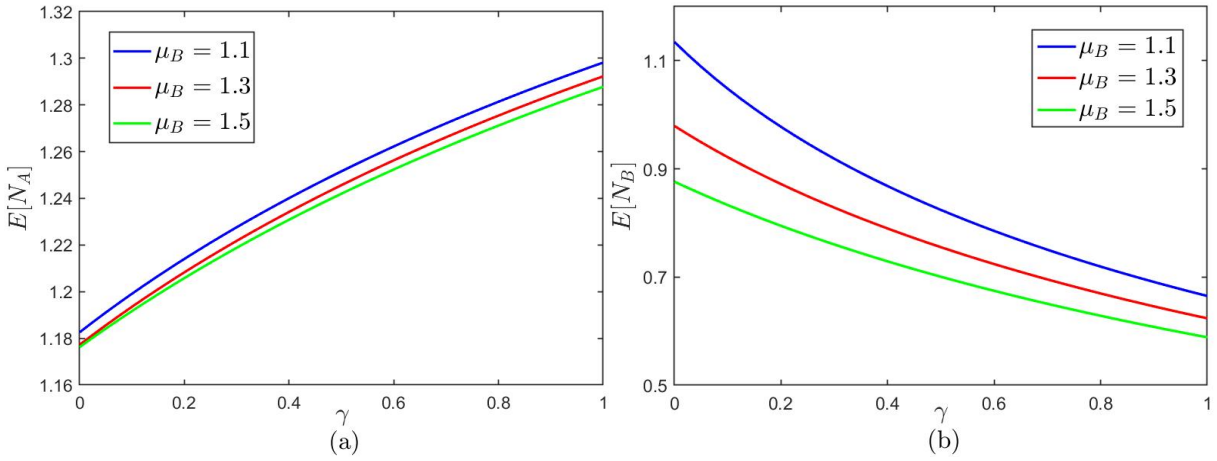

Figure 7. The impact of γ on the average number of COVID-19 patients.

Figure 7 shows the trend in the average number of symptomatic patients and asymptomatic

patients when the transition rate from asymptomatic patients to symptomatic patients (γ) increases. The blue, red and green lines in Figure 7 represent the cases where the disappearance rate of asymptomatic patients (μ_B) is given as 1.1, 1.3 and 1.5, respectively. Each line in Figure 7(a) indicates that the average number of symptomatic patients increases as γ increases. Furthermore, comparing the three lines reveals that the larger the value of μ_B, the smaller the average number of symptomatic patients. In contrast, Figure 7(b) demonstrates that the average number of asymptomatic patients decreases as γ and μ_B increase.

According to the results in Figure 7, increasing the transition rate from asymptomatic patients to symptomatic patients leads to an increase in the average number of symptomatic patients and a decrease in the average number of asymptomatic patients. Due to the higher infection rate of symptomatic patients compared to asymptomatic patients, and the longer duration of infection caused by asymptomatic patients, the impact of increasing the transition rate on controlling the transmission of infectious diseases may vary depending on the specific circumstances.

4) Case 4: Let's take μ_A as 5, 6 and 7, respectively, and observe the impact of μ_B varying in the interval [0, 1] on $E[N_A]$ and $E[N_B]$. The numerical results are shown in Figure 8.

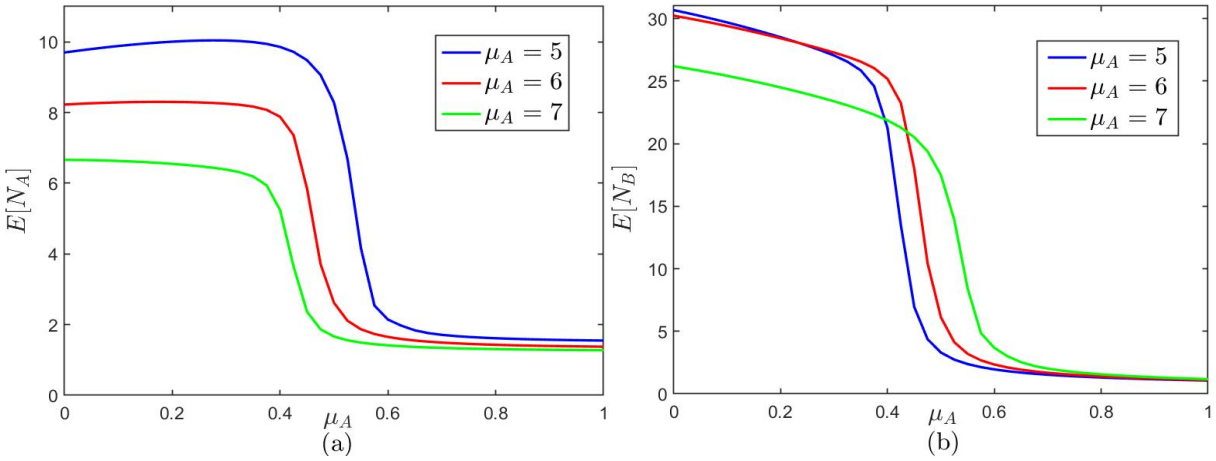

Figure 8. The impact of μ_B on the average number of COVID-19 patients.

Figure 8 illustrates the trend in the average number of symptomatic patients and asymptomatic patients as the disappearance rate of asymptomatic patients (μ_B) increases. The blue, red and green lines in Figure 8 represent the cases in which the disappearance rate of asymptomatic patients (μ_A) is given as 5, 6 and 7, respectively. Each line in Figure 8(a) shows that the average number of symptomatic patients decreases as μ_B increases. The same trend is observed in Figure 8(b), where the average number of asymptomatic patients decreases as μ_B increases. Furthermore, the decline in the average number of symptomatic and asymptomatic patients is notably faster within the range of 0.4–0.6 compared to other values.

Based on the findings in Figure 8, increasing the disappearance rate of asymptomatic patients results in a decline in the average number of symptomatic and asymptomatic patients. Notably, there exists a specific range where increasing the disappearance rate of asymptomatic patients can lead to a more substantial reduction in the average number of patients compared to other scenarios. Therefore, it is advisable to identify this range prior to implementing non-drug interventions, as it can contribute

to more cost-effective control of the infectious disease transmission process.

5) Case 5: In order to examine the impact of non-drug intervention strategies and vaccines on infectious disease transmission, we introduce two variables: w and v, representing the effects of non-drug interventions and drug interventions (e.g., vaccines) on the infection rate of disease, respectively. Additionally, let β_1 and β_2 be the weights representing the effects of non-drug interventions and drug interventions on the infection rate of infectious disease, respectively. Obviously, $\beta_1 + \beta_2 = 1$. Based on this, we have

$$\lambda' = (\beta_1 \frac{1}{w} + \beta_2 \frac{1}{v})\lambda \tag{51}$$

where λ' denotes the infection rate considering the impact of both non-drug interventions and drug interventions, and λ denotes the infection rate without considering them. In this situation, we set the benchmark parameter value for the infection rate without considering the impact of non-drug interventions and drug interventions. Under different situations, we examine the impact of v on the COVID-19 transmission in three cases: $\beta_1 = 0.5$, $\beta_2 = 0.5$; $\beta_1 = 0.2$, $\beta_2 = 0.8$; and $\beta_1 = 0.8$, $\beta_2 = 0.2$.

Figure 9 gives the impact of v on the $E[N_A]$ and $E[N_B]$ under different w in various weight combination cases. The blue, red and green lines in Figure 9 represent the cases where the effect of non-drug interventions on the infection rate of disease (w) is set as 1, 1.5 and 2, respectively. Each line in Figure 9(a) shows that the average number of symptomatic patients decreases as v increases. Furthermore, comparing the three lines reveals that the average number of symptomatic patients decreases as w increases. The same trend is observed in Figure 9(b), where the average number of asymptomatic patients also decreases as v and w increase. According to the results in Figure 9, both non-drug interventions and drug interventions contribute to a decrease in the average number of symptomatic and asymptomatic patients, thus effectively controlling the transmission of infectious disease.

Based on the stability and the numerical analysis, we can know that the infectious disease transmission process with asymptomatic patients is stable when $\lambda_{AA} < \mu_A$. Controlling the infection rates and disappearance rates of symptomatic and asymptomatic patients can affect the average number of symptomatic and asymptomatic patients when this infectious disease transmission process is stable. Therefore, while paying attention to the control of symptomatic patients, the screening and control of asymptomatic patients should not be neglected. Specifically, the average number of symptomatic patients increases with λ_{AA}, λ_{AB}, λ_{BA}, λ_{BB} and γ; and decreases with μ_A and μ_B. The average number of asymptomatic patients increases with λ_{AA}, λ_{AB}, λ_{BA} and λ_{BB}; and decreases with γ, μ_A and μ_B. Therefore, if the control strategies can decrease the parameters λ_{AA}, λ_{AB}, λ_{BA} and λ_{BB} or increase the parameters μ_A and μ_B, it can be helpful for the scientific control and management of the epidemic. Increasing γ is effective for controlling transmission caused by asymptomatic patients, but not for transmission caused by symptomatic patients. Furthermore, there exists an optimal interval in which increasing μ_B can more effectively control the transmission of infectious diseases. Therefore, decisions on how to control γ and μ_B should be made based on the specific situation. Both non-drug interventions and drug interventions can influence the parameters of the infectious disease transmission. The above findings are in line with those in the existing related literature [26–30]. In addition, we consider the impact of transmission from asymptomatic patients on the system. Based on the research results of this paper and the specific situation of the region, it is

recommended to regulate the intensity of infectious disease control strategies and the level of drug interventions at an optimal level to achieve effective and cost-efficient control of infectious diseases.

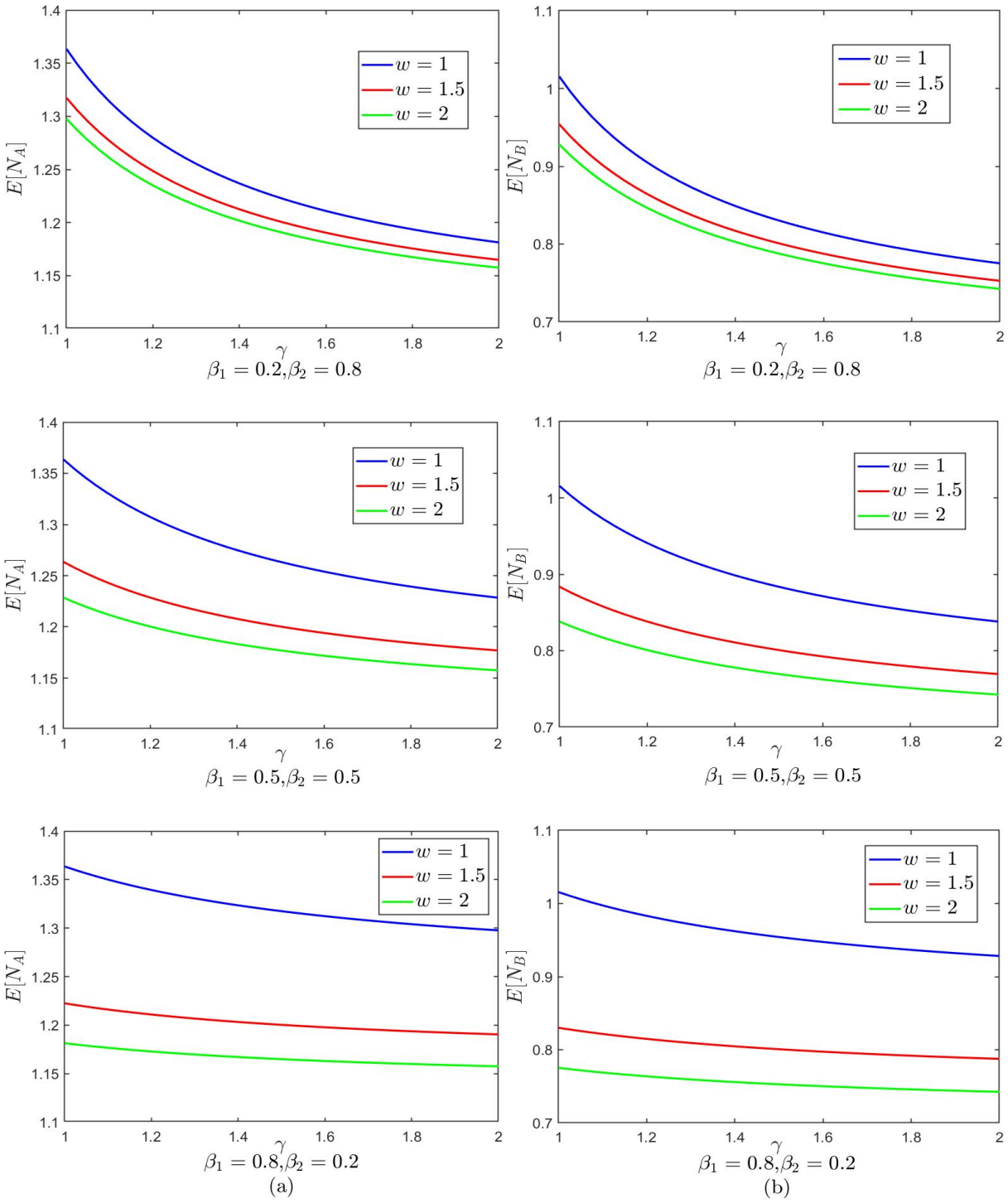

Figure 9. The impact of v on the average number of COVID-19 patients.

7. Conclusions and future research

In this paper, we developed a new Markov process of infectious disease with infinite-state space to study the stable condition of an infectious disease system, which is based on the continuous time

level-dependent QBD process. Compared to existing studies, we considered the long-term impact of asymptomatic patients on the transmission process of infectious diseases. We gave a simple sufficient condition for the stability of the model by using the mean drift technique. We obtained the stationary probability vector by means of RG-factorizations. Using this stationary probability vector, we gave some important performance measures of this model. Finally, we verified the feasibility of this paper by using some numerical experiments through taking COVID-19 as an example. The effects of some key parameters on the stationary probability of the model and the role of two types of patients on the transmission of infectious diseases were also quantitatively analyzed based on performance analysis. The modeling technique proposed in this paper is operational and general. It can be applied to other infectious diseases if they consist of symptomatic and asymptomatic patients and both types of patients can infect and generate the two types of patients. Therefore, the methodology and results of this paper can provide some useful theoretical basis and technical support for the long-term scientific prevention and control of infectious diseases.

In future studies, the proposed model in this paper can be extended in the following ways:

1) In the framework of the homogeneous Markov process, the patient infection and disappearance processes can be extended from Poisson processes to Markovian arrival processes (MAPS), whose purpose is to reflect the burstiness and short-time clustering characteristics of infectious diseases.

2) Extending the patient infection and treatment process from a homogeneous Poisson process to a non-homogeneous Poisson process, i.e., the parameters can vary over time.

3) Develop a data-driven Markov process by deriving the infection, cure and death rates of patients in the model based on the actual data of infectious diseases.

4) Apply Markov decision process to study optimal control strategies in infectious disease prevention and control, e.g., vaccine input strategy, isolation intensity strategy, economic strategy and etc.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

Data availability

The data used in numerical experiments have been uploaded to the database of Zenodo (https://doi.org/10.5281/zenodo.8280820).

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Conflict of interest

The authors declare there is no conflict of interest.

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Appendix

In this appendix, we use the mean drift technique to provide a simple sufficient condition for the stability of the QBD process. Ensuring model stability is essential for the analysis of the model.

Theorem A1. *When* $\mu_A > \lambda_{AA}$*, this QBD process of infectious disease* (Q) *is irreducible and positive recurrent. Thus, the QBD process of infectious disease is stable.*

Proof. Let $\mathbf{A}_k = A_{k,k-1} + A_{k,k} + A_{k,k+1}$, where k denotes the k th level, $k \geq 1$. Let $h_i = k\lambda_{AB} + i(\gamma + \mu_B + \lambda_{BB})$, $o_i = k\lambda_{AB} + i\lambda_{BB}$. Then, $\mathbf{A}_k$ can be written as follows:

$$\mathbf{A}_k = \begin{pmatrix} -h_0 & o_0 & & & & & \\ \gamma+\mu_B & -h_1 & o_1 & & & & \\ & 2(\gamma+\mu_B) & -h_2 & o_2 & & & \\ & & \ddots & \ddots & \ddots & & \\ & & & (M-1)(\gamma+\mu_B) & -h_{M-1} & o_{M-1} & \\ & & & & M(\gamma+\mu_B) & -h_M & \end{pmatrix}.$$

Note that it is easy to see that $\mathbf{A}_k$ is a finite state and irreducible birth-death process, so this process must be positive recurrent and stable. Therefore, this birth-death process $\mathbf{A}_k$ has the stable state and stationary probability vector.

Let $\boldsymbol{\alpha}^{(k)} = (\alpha_0^{(k)}, \alpha_1^{(k)}, \alpha_2^{(k)}, \ldots, \alpha_M^{(k)})$ be the stationary probability vector of $\mathbf{A}_k$. Then, from the characteristics of the stationary probability vector, we can obtain

$$\boldsymbol{\alpha}^{(k)}\mathbf{A}_k = \boldsymbol{\alpha}^{(k)}(A_{k,k-1} + A_{k,k} + A_{k,k+1}) = 0, \tag{A1}$$

$$\boldsymbol{\alpha}^{(k)}\mathbf{e} = 1, \tag{A2}$$

where $\mathbf{e}$ is a column vector of ones with a suitable size.

According to Eq (A1), we have

$$\begin{aligned} \boldsymbol{\alpha}^{(k)}\mathbf{A}_k = \big(&\alpha_0^{(k)}(-k\lambda_{AB}) + \alpha_1^{(k)}(\gamma+\mu_B), \alpha_0^{(k)}k\lambda_{AB} + \alpha_1^{(k)}\left[-k\lambda_{AB} - (\mu_B+\gamma+\lambda_{BB})\right] + \alpha_2^{(k)}2(\gamma+\mu_B), \\ &\alpha_1^{(k)}(k\lambda_{AB}+\lambda_{BB}) + \alpha_2^{(k)}\left[-k\lambda_{AB} - 2(\mu_B+\gamma+\lambda_{BB})\right] + \alpha_3^{(k)}3(\gamma+\mu_B), \ldots, \\ &\alpha_{M-2}^{(k)}\left[k\lambda_{AB} + (M-2)\lambda_{BB}\right] + \alpha_{M-1}^{(k)}\left[-k\lambda_{AB} - (M-1)(\mu_B+\gamma+\lambda_{BB})\right] + \alpha_M^{(k)}M(\gamma+\mu_B), \\ &\alpha_{M-1}^{(k)}\left[k\lambda_{AB} + (M-1)\lambda_{BB}\right] + \alpha_M^{(k)}\left[-k\lambda_{AB} - M(\mu_B+\gamma+\lambda_{BB})\right]\big). \end{aligned} \tag{A3}$$

From Eq (A1) and the first term in Eq (A3), we have

$$\alpha_0^{(k)}(-k\lambda_{AB}) + \alpha_1^{(k)}(\gamma+\mu_B) = 0,$$

$$\alpha_1^{(k)} = \frac{k\lambda_{AB}}{\gamma+\mu_B}\alpha_0^{(k)}. \tag{A4}$$

From Eq (A1), the second term in Eqs (A3) and (A4), we have

$$\alpha_0^{(k)}k\lambda_{AB} + \alpha_1^{(k)}\left[-k\lambda_{AB} - (\mu_B+\gamma+\lambda_{BB})\right] + \alpha_2^{(k)}2(\gamma+\mu_B) = 0,$$

$$\alpha_2^{(k)} = \frac{k\lambda_{AB}(k\lambda_{AB}+\lambda_{BB})}{2!(\gamma+\mu_B)^2}\alpha_0^{(k)}.$$

Similarly, we can obtain Eqs (A5) and (A6) to get $\alpha_l^{(k)}$.

$$\alpha_{l-2}^{(k)}\left(k\lambda_{AB} + (l-2)\lambda_{BB}\right) + \alpha_{l-1}^{(k)}\left[-k\lambda_{AB} - (l-1)(\mu_B+\gamma+\lambda_{BB})\right] + \alpha_l^{(k)}l(\gamma+\mu_B) = 0, l \geq 1, \tag{A5}$$

$$\alpha_l^{(k)} = \frac{\prod_{i=0}^{l-1}(k\lambda_{AB}+i\lambda_{BB})}{l!(\gamma+\mu_B)^l}\alpha_0^{(k)}, l \geq 1. \tag{A6}$$

After that, depending on Eqs (A2) and (A6), we can obtain

$$\alpha_0^{(k)}+\frac{k\lambda_{AB}}{\gamma+\mu_B}\alpha_0^{(k)}+\frac{\prod_{i=0}^{l-1}(k\lambda_{AB}+i\lambda_{BB})}{2!(\gamma+\mu_B)^2}\alpha_0^{(k)}+\cdots+\frac{\prod_{i=0}^{M-1}(k\lambda_{AB}+i\lambda_{BB})}{M!(\gamma+\mu_B)^M}\alpha_0^{(k)}=1,$$

$$\alpha_0^{(k)}=\frac{1}{1+\sum_{j=1}^{M}\frac{\prod_{i=0}^{j-1}(k\lambda_{AB}+i\lambda_{BB})}{j!(\gamma+\mu_B)^j}},l\geq 1.$$

Using the aforementioned description, we can obtain the stationary probability vector $\boldsymbol{\alpha}^{(k)}$ for the birth-death process $\mathbf{A}_k$. Subsequently, we can leverage this vector to discuss the stable conditions of the QBD process proposed in this paper. In particular, we analyze two key rates when discussing the stable state: the upward mean drift rate and the downward mean drift rate.

The upward mean drift rate: mean drift rate from level k to level $k+1$

$$\begin{aligned}\boldsymbol{\alpha}^{(k)}A_{k,k+1}\mathbf{e}&=\boldsymbol{\alpha}^{(k)}(k\lambda_{AA},k\lambda_{AA}+(\lambda_{BA}+\gamma),\ldots,k\lambda_{AA}+M(\lambda_{BA}+\gamma))^T\\&=\alpha_0^{(k)}k\lambda_{AA}+\alpha_1^{(k)}\left[k\lambda_{AA}+(\lambda_{BA}+\gamma)\right]+\cdots+\alpha_M^{(k)}\left[k\lambda_{AA}+M(\lambda_{BA}+\gamma)\right]\\&=k\lambda_{AA}(\alpha_0^{(k)}+\alpha_0^{(k)}+\cdots\alpha_0^{(k)})+\left[\alpha_1^{(k)}(\lambda_{BA}+\gamma)+\alpha_2^{(k)}2(\lambda_{BA}+\gamma)+\cdots+\alpha_M^{(k)}M(\lambda_{BA}+\gamma)\right]\\&=k\lambda_{AA}+\left[\alpha_1^{(k)}(\lambda_{BA}+\gamma)+\alpha_2^{(k)}2(\lambda_{BA}+\gamma)+\cdots+\alpha_M^{(k)}M(\lambda_{BA}+\gamma)\right].\end{aligned}$$

The downward mean drift rate: mean drift rate from level k to level $k-1$

$$\begin{aligned}\boldsymbol{\alpha}^{(k)}A_{k,k-1}\mathbf{e}&=\boldsymbol{\alpha}^{(k)}(k\mu_A,k\mu_A,\ldots,k\mu_A)^T\\&=\alpha_0^{(k)}k\mu_A+\alpha_1^{(k)}k\mu_A+\cdots+\alpha_M^{(k)}k\mu_A\\&=k\mu_A(\alpha_0^{(k)}+\alpha_0^{(k)}+\cdots\alpha_0^{(k)})\\&=k\mu_A.\end{aligned}\tag{A7}$$

According to the mean drift technique, a QBD process is positive recurrent if its downward mean drift rate is greater than its upward mean drift rate. Therefore, we proceed to calculate

$$\begin{aligned}\boldsymbol{\alpha}^{(k)}A_{k,k-1}\mathbf{e}-\boldsymbol{\alpha}^{(k)}A_{k,k+1}\mathbf{e}&=k\mu_A-k\lambda_{AA}+\left[\alpha_1^{(k)}(\lambda_{BA}+\gamma)+\alpha_2^{(k)}2(\lambda_{BA}+\gamma)+\cdots+\alpha_M^{(k)}M(\lambda_{BA}+\gamma)\right]\\&=k(\mu_A-\lambda_{AA})+\left[\alpha_1^{(k)}(\lambda_{BA}+\gamma)+\alpha_2^{(k)}2(\lambda_{BA}+\gamma)+\cdots+\alpha_M^{(k)}M(\lambda_{BA}+\gamma)\right].\end{aligned}\tag{A8}$$

In Eq (A8), M is a fixed value, so $\alpha_1^{(k)}(\lambda_{BA}+\gamma)+\alpha_2^{(k)}2(\lambda_{BA}+\gamma)+\cdots+\alpha_M^{(k)}M(\lambda_{BA}+\gamma)$ is a finite value. If $\mu_A-\lambda_{AA}>0$ and k tends to positive infinity, $k(\mu_A-\lambda_{AA})$ will tends to positive infinity. Therefore, when k tends to positive infinity, Eq (A8) must be larger than 0. This means that the downward mean drift rate of the QBD process Q is greater than its upward mean drift rate. Therefore, Q is positive recurrent and stable. This completes the proof.