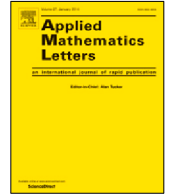




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On the dynamical model for COVID-19 with vaccination and time-delay effects: A model analysis supported by Yangzhou epidemic in 2021



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ABSTRACT

The 2019 novel coronavirus (COVID-19) emerged at the end of 2019 has a great influence on the health and lives of people all over the world. The spread principle is still unclear. This paper considers a novel evolution model of COVID-19 in terms of an integral–differential equation, involving vaccination effect and the incubation of COVID-19. The proposed mathematical model is rigorously analyzed on its asymptotic behavior with new probability functions, showing the final spread tendency. Moreover, our model is also verified numerically by the practical epidemic data of COVID-19 in Yangzhou from July to August 2021.

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1. Introduction

In March 2020, the World Health Organization declared the COVID-19 outbreak to be a global pandemic. With the active cooperations of countries around the world, the epidemic is under control temporarily, especially due to the invention of 2019-nCoV vaccine. However, a new round of epidemic of the Delta COVID-19 variants broke out again in Nanjing on July 20, 2021, caused by the external input from a Russian flight. Then the epidemic spread out quickly, leading to three serious epidemic sites in China: Nanjing, Zhangjiajie and Yangzhou. Noticing that the 2019-nCoV vaccine has been injected to most of the people in China and some serious measurements have been taken to control the disease, it is necessary to restudy the COVID-19 virus, including the vaccination effect and its propagation mechanism.

Except for some well-known models on general epidemic diseases such as Logistic model, SIS model, SIR model and SEIR model [1,2], there already have been extensive works on the propagation models for

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COVID-19 epidemic, with the aim to overcome the shortcomings of these classical models ignoring either the human mobility or the lag effect of the virus incubation. In fact, it is reported that the epidemic outbreak in Yangzhou in July 2021 comes from the turnover of an “old lady” within the city who traveled to Yangzhou from Nanjing.

Some existing studies have carefully considered the influence of imported patients, isolation of infected patients, incubation period of the disease, cure ratio and cure time of patients, as well as mortality of patients. Especially some dynamic models of COVID-19 considering the time-delay effect have been derived, see [3–6]. However, these models do not consider the impact of vaccination. On the other hand, in the evolution models involving the time-delay effects of epidemic infections, the probability functions for incubation period should be assumed artificially, we also need to consider other probability functions theoretically and numerically.

As a new but typical epidemic disease, the spread of COVID-19 depends on:

- The harmful nature of the viruses themselves, such as the incubation period, infection ratio and recovery ratio;
- The cognition and therapeutic effects of contemporary medicine on viruses, including the cure period, cure time of patients and the effectiveness of vaccine;
- Measures to control the viruses spread, such as travel restrictions, isolating patients, controlling the imported cases and vaccination ratios.

In this paper, we consider the interactions of isolation effect, external input, recovery ratio and vaccination on the spread of the epidemic comprehensively. Different from the existing model proposed in [3], we consider new density functions obeyed by the incubation period and cure period, together with the vaccination effects in the infection model. We theoretically proved that, for this novel model, the epidemic can be finally controlled, i.e., the infected patients will eventually disappear by adequate isolation and high vaccination ratio. Our work shows that the model can effectively describe the propagation mechanism of infectious diseases with long latency, and the numerical results reflect the characteristics of viruses and vaccines in predicting the epidemic tendency in different periods.

This paper is organized as follows. In Section 2, we establish a novel model for COVID-19 introducing new probability functions and the vaccination effect comprehensively. Then the asymptotic behavior of the number of the infected people is rigorously proved, together with the numerical verifications of the proposed model in terms of the real epidemic data of Yangzhou in July 2021 in Section 3, showing the validity of the proposed model.

2. Transmission model with vaccination effects

Suppose there exist some infected patients at initial time. Then all the infected patients, assumed to be of either mild symptoms or severe ones, are generated by the spread of local patients and external input cases. However, we should consider vaccination effects decreasing the infection ratio. Denote by $I_s(t)$ the cumulative number of the infected persons at time interval $[0, t]$. Similarly to the model proposed in [3], we introduce the following quantities:

- $s(t)$: imported cases at time t (suppose they are just infected);
- $i(t)$: newly infected people due to internal infection at time t ;
- $j(t)$: newly confirmed people at time t ;
- $c(t)$: cured persons with mild symptoms at time t , including people being vaccinated $c_1(t)$ and those not being vaccinated $c_2(t)$;
- $d(t)$: cured or died persons with severe symptoms at time t , including people being vaccinated $d_1(t)$ and those not being vaccinated $d_2(t)$.

Our preliminary aim is to propose a novel nonlocal model with rigorous asymptotic analysis, and to show the validity of this model if the parameters such as $d(t)$ in this model can be specified appropriately. However, the influence of the parameters cannot be verified numerically if the epidemic period is short such as the Yangzhou cases.

Such a new configuration is more complicated, since even if for people who have been vaccinated, they can still be the sources of infections due to the possible invalidity of the vaccines for special individuals. To establish the propagation law of epidemic by infection process and medical treatments, we assume that the latent period, cure period of the diseases are random variables represented by some density functions. More precisely, we assume

- All the random variables τ describing the propagation indices of COVID-19 in our model are independently and identically distributed (IID) for different individuals, with the probability density function $h_s(\tau; \tau_s)$ and average value τ_s ;
- The infection ratio $\beta > 0$ of the disease is a constant;
- The patients are no longer of infectivity once they are isolated, cured or died.

The vaccination plays an important role in two ways. Firstly, the vaccinated persons have less proportion to be infected compared with the non-vaccinated ones. Secondly, the vaccinated people, even if they get infected, will be of lower death ratio compared with the non-vaccinated ones. For time $t > 0$, introduce $\kappa_1(t) \in (0, 1)$ the isolation ratio for the infected people, $\kappa_2(t) \in (0, 1)$ the vaccination ratio of the population, and $\kappa_3(t) \in (0, 1)$ the ratio of effectiveness of vaccination. Then the newly infected people $i(t)$ fall into two parts: persons who have been vaccinated but ineffective and those who have not been vaccinated, i.e., we have the representation

$$i(t) = i_1(t) + i_2(t) = \beta(1 - \kappa_1(t))\kappa_2(t)(1 - \kappa_3(t))I_s(t) + \beta(1 - \kappa_1(t))(1 - \kappa_2(t))I_s(t). \tag{2.1}$$

To describe the infection propagation from the infected people at initial time, namely, $I_s(0) = I_s^0$, we consider it equivalently as a continuous and uniform input $s_0(t)$ in the time interval $[0, \epsilon_0]$ for small $\epsilon_0 > 0$. Then the infection source $S(t) = s(t) + s_0(t)$, where

$$s_0(t) = \begin{cases} \frac{2}{\epsilon_0^2} I_s^0 (\epsilon_0 - t), & t \in [0, \epsilon_0], \\ 0, & t > \epsilon_0 \end{cases} \tag{2.2}$$

satisfying $\int_0^{\epsilon_0} s_0(t)dt = I_s^0$. We also decompose

$$S(t) \equiv \frac{\kappa_2(t)(1 - \kappa_3(t))}{1 - \kappa_2(t)\kappa_3(t)}S(t) + \frac{1 - \kappa_2(t)}{1 - \kappa_2(t)\kappa_3(t)}S(t) := S_1(t) + S_2(t),$$

where $S_1(t)$ is those who have been vaccinated but non-effective, while $S_2(t)$ is those who have not been vaccinated.

All the newly infected people $i(t) + S(t)$ at time t will get confirmed once they have passed the incubation period τ with average τ_j , which is a random variable with the density function $h_j(\tau; \tau_j)$. Similarly to the classification of newly infected people, the newly confirmed cases $j(t)$ can also be divided into two components, i.e.,

$$j(t) = \int_0^t h_j(t - \tau; \tau_j) \sum_{k=1}^2 [i_k(\tau) + S_k(\tau)]d\tau := j_1(t) + j_2(t). \tag{2.3}$$

Let $p_1, p_2 \in (0, 1)$ be the ratios of patients with mild symptoms for vaccinated patients and non-vaccinated ones, respectively. Corresponding to $j_k(t)$ with $k = 1, 2$, we describe the cured effects for patients with mild symptoms by the kernel functions $h_{c_k}(\tau; \tau_{c_k})$, while the cured and dead effects for patients with severe

symptoms are described by kernel functions $h_{d_k}(\tau; \tau_{d_k})$, where τ is the random variable with average τ_{c_k}, τ_{d_k} for the performances of medical treatments. Then we have

$$c(t) = \sum_{k=1}^2 c_k(t) = p_1 \int_0^t h_{c_1}(\tau; \tau_{c_1}) j_1(t - \tau) d\tau + p_2 \int_0^t h_{c_2}(\tau; \tau_{c_2}) j_2(t - \tau) d\tau, \tag{2.4}$$

$$d(t) = \sum_{k=1}^2 d_k(t) = (1 - p_1) \int_0^t h_{d_1}(\tau; \tau_{d_1}) j_1(t - \tau) d\tau + (1 - p_2) \int_0^t h_{d_2}(\tau; \tau_{d_2}) j_2(t - \tau) d\tau. \tag{2.5}$$

Obviously, there should be the relations $\tau_{c_1} \leq \tau_{c_2}, p_1 \geq p_2$, revealing the effect of vaccination. Using the balance equation

$$I'_s(t) = s(t) + i(t) - c(t) - d(t) \tag{2.6}$$

and the above analysis, we obtain an integral–differential system with respect to $I_s(t)$:

$$\begin{aligned} I'_s(t) = & s(t) + \beta(1 - \kappa_1(t))\kappa_2(t)(1 - \kappa_3(t))I_s(t) + \beta(1 - \kappa_1(t))(1 - \kappa_2(t))I_s(t) - \\ & \int_0^t \beta(1 - \kappa_1(z))\kappa_2(z)(1 - \kappa_3(z))I_s(z) \\ & \int_z^t [p_1 h_{c_1}(t - \tau; \tau_{c_1}) + (1 - p_1)h_{d_1}(t - \tau; \tau_{d_1})] h_j(\tau - z; \tau_j) d\tau dz - \\ & \int_0^t \beta(1 - \kappa_1(z))(1 - \kappa_2(z))I_s(z) \\ & \int_z^t [p_2 h_{c_2}(t - \tau; \tau_{c_2}) + (1 - p_2)h_{d_2}(t - \tau; \tau_{d_2})] h_j(\tau - z; \tau_j) d\tau dz - \\ & \int_0^t S_1(z) \int_z^t [p_1 h_{c_1}(t - \tau; \tau_{c_1}) + (1 - p_1)h_{d_1}(t - \tau; \tau_{d_1})] h_j(\tau - z; \tau_j) d\tau dz - \\ & \int_0^t S_2(z) \int_z^t [p_2 h_{c_2}(t - \tau; \tau_{c_2}) + (1 - p_2)h_{d_2}(t - \tau; \tau_{d_2})] h_j(\tau - z; \tau_j) d\tau dz, \end{aligned} \tag{2.7}$$

together with the initial value

$$I_s(0) = I_s^0. \tag{2.8}$$

Once we solve $I_s(t)$ from (2.7)–(2.8), $(i(t), j(t), c(t), d(t))$ can then be determined, which means that all the relevant quantities describing the spread of COVID-19 can be obtained.

Remark 2.1. Different from our model in [3], where we remove the infected people in terms of the cured persons $c(t)$ and died persons $d(t)$, here we establish the relation (2.6), where $c(t)$ represents the number of cured persons of mild symptoms, while $d(t)$ is the number of cured and died persons of severe symptoms.

3. Asymptotic behavior of the dynamical model

Instead of the Gaussian and Weibull distributions for random variables in our previous work [3], here we assume that all the above random variables are of the Gamma distributions, i.e., for the Gamma function

$$f(t; \alpha, \lambda) = \begin{cases} \frac{\lambda^\alpha}{\Gamma(\alpha)} t^{\alpha-1} e^{-\lambda t}, & t \geq 0, \\ 0, & t < 0 \end{cases} \tag{3.1}$$

with $\alpha, \lambda > 0$ the shape parameters, we take

$$h(t; \tau, \lambda) = f(t; \tau\lambda, \lambda) \tag{3.2}$$

for different values (τ, λ) as the density functions in our model. It is easy to see that the random variable t obeying the density function $h(t; \tau, \lambda)$ is of the average τ for any $\lambda > 0$. The mathematical models and the numerical simulations with other density functions are the same, but the theoretical analysis depends on the form of density function and then needs to be carried out separately.

Since the specification of the distribution function is artificial, the purpose we take Gamma distribution here is to show the model is stable with respect to the forms of distribution functions. We would like to emphasize that the Gamma distribution is also applied to recover the reproduction number for COVID-19, see [7].

In general, the epidemic situation should tend to some stable status after a long time. Any reasonable mathematical model describing the spread of COVID-19 should satisfy such a requirement. We will show this feature for our proposed model, provided that some *a-priori* assumptions on isolation ratio, imported cases and the vaccination effect be specified.

Theorem 3.1. *For any $T > 0$, there exists a unique solution $I_s(t) \in C[0, T]$ to (2.7)–(2.8). Assume that all the random variables describing the disease properties obey the Gamma distribution (3.2), and the imported cases $s(t)$ satisfies the growing condition*

$$\int_0^\infty s(t)e^{\frac{\lambda}{2}t} dt \leq s^* < \infty. \tag{3.3}$$

Then for $\beta\kappa_0 \in (0, 1)$ small enough with $\kappa_0 := \max_{[0, \infty)}(1 - \kappa_1(t))(1 - \kappa_2(t)\kappa_3(t))$, we have

$$\lim_{t \rightarrow \infty} I_s(t) = 0, \quad \lim_{t \rightarrow \infty} c(t) = 0, \quad \lim_{t \rightarrow \infty} d(t) = 0. \tag{3.4}$$

Proof. The unique existence of $I_s(t)$ to (2.7)–(2.8) comes from the solvability of the linear Volterra integral equation of the second kind with continuous kernel. For $k = 1, 2$, define

$$M_k(t) := [p_k h_{c_k}(t; \tau_{c_k}) + (1 - p_k)h_{d_k}(t; \tau_{d_k})] * h_j(t; \tau_j) := p_k h_{j c_k}(t) + (1 - p_k)h_{j d_k}(t).$$

By integrating (2.7) in $[0, T]$ and exchanging the order of integrations, we get

$$I_s(t) = I_s^0 + \int_0^t S(z)M_S(t - z, z)dz + \beta \int_0^t (1 - \kappa_1(z))I_s(z)M_I(t - z, z)dz - \int_0^t s_0(z)dz, \tag{3.5}$$

where

$$\begin{cases} M_S(y, z) := 1 - \frac{\kappa_2(z)(1 - \kappa_3(z))}{1 - \kappa_2(z)\kappa_3(z)} \int_0^y M_1(\tau) d\tau - \frac{1 - \kappa_2(z)}{1 - \kappa_2(z)\kappa_3(z)} \int_0^y M_2(\tau) d\tau, \\ M_I(y, z) := 1 - \kappa_2(z)\kappa_3(z) - \kappa_2(z)(1 - \kappa_3(z)) \int_0^y M_1(\tau) d\tau - (1 - \kappa_2(z)) \int_0^y M_2(\tau) d\tau. \end{cases}$$

Since $\{h_i(t; \tau_i), i = j, c_1, c_2, d_1, d_2\}$ are of the form of Gamma function (3.2) of (τ_i, λ) , $(i = j, c_1, c_2, d_1, d_2)$ for any specified $\lambda > 0$, we have from the straightforward computations that

$$\begin{aligned} \int_0^{t-z} M_k(\tau) d\tau &= \int_0^{t-z} [p_k h_{j c_k}(\tau) + (1 - p_k)h_{j d_k}(\tau)] d\tau \\ &= 1 - p_k \int_{t-z}^{+\infty} \gamma_{j c_k} \tau^{\lambda \tau_{j c_k} - 1} e^{-\lambda \tau} d\tau - (1 - p_k) \int_{t-z}^{+\infty} \gamma_{j d_k} \tau^{\lambda \tau_{j d_k} - 1} e^{-\lambda \tau} d\tau, \end{aligned}$$

where $\tau_{jm} = \tau_j + \tau_m$, $\gamma_{jm} = \frac{\lambda^{\lambda \tau_{jm}}}{\Gamma(\lambda \tau_{jm})}$ for $m = c_k, d_k$ and $k = 1, 2$. For $\lambda \tau_{jm} - 1 > 0$, there exists some constant $c(\tau_{jm}, \lambda) > 0$ such that $\gamma_{jm} \tau^{\lambda \tau_{jm} - 1} \leq c(\tau_{jm}, \lambda) e^{\frac{\lambda}{2} \tau}$ for $\tau \in [0, +\infty)$ with $m = c_k, d_k$ and $k = 1, 2$. So we have

$$\int_{t-z}^{+\infty} \gamma_{jm} \tau^{\lambda \tau_{jm} - 1} e^{-\lambda \tau} d\tau \leq \int_{t-z}^{+\infty} c(\tau_{jm}, \lambda) e^{-\frac{\lambda}{2} \tau} d\tau = \frac{2c(\tau_{jm}, \lambda)}{\lambda} e^{-\frac{\lambda}{2}(t-z)} \leq \frac{2c_0}{\lambda} e^{-\frac{\lambda}{2}(t-z)}$$

Table 1
Comparisons between real and simulant data in Yangzhou by our model.

date	07/28	07/29	07/30	07/31	08/01	08/02	08/03
$j_e(t)(j_t(t))$	2(2)	4(0)	10(2)	12(9)	26(20)	40(33)	32(45)
$J_e(t)(J_t(t))$	2(2)	6(2)	16(4)	28(13)	54(33)	94(66)	126(111)
date	08/04	08/05	08/06	08/07	08/08	08/09	08/10
$j_e(t)(j_t(t))$	36(53)	58(57)	52(56)	36(52)	38(47)	48(40)	54(33)
$J_e(t)(J_t(t))$	162(164)	220(221)	272(277)	308(329)	346(376)	394(416)	448(449)
date	08/11	08/12	08/13	08/14	08/15	08/16	08/17
$j_e(t)(j_t(t))$	37(27)	25(21)	18(17)	18(13)	6(9)	3(7)	6(5)
$J_e(t)(J_t(t))$	485(476)	510(497)	528(514)	546(527)	552(536)	555(543)	561(548)
date	08/18	08/19	08/20	08/21	08/22	08/23	
$j_e(t)(j_t(t))$	3(4)	2(3)	1(2)	1(1)	0(1)	0(1)	
$J_e(t)(J_t(t))$	564(552)	566(555)	567(557)	568(558)	568(559)	568(560)	

with $c_0 := \max\{c(\tau_{jm}, \lambda), m = c_1, c_2, d_1, d_2\}$. This uniform bound says

$$0 < M_S(t - z, z) \leq \frac{2c_0}{\lambda} e^{-\frac{\lambda}{2}(t-z)}, \quad 0 < M_I(t - z, z) \leq (1 - \kappa_2(z)\kappa_3(z)) \frac{2c_0}{\lambda} e^{-\frac{\lambda}{2}(t-z)}. \tag{3.6}$$

By (3.6), we can estimate the integrals in (3.5). Since $\text{supp}s_0(t) = [0, \epsilon_0]$ and $\int_0^{\epsilon_0} s_0(z)dz = I_s^0$, (3.5) finally generates

$$\begin{aligned} I_s(t)e^{\frac{\lambda t}{2}} &\leq \frac{2c_0}{\lambda} \int_0^{\epsilon_0} s_0(z)e^{\frac{\lambda z}{2}} dz + \frac{2c_0}{\lambda} \int_0^t s(z)e^{\frac{\lambda z}{2}} dz + \frac{2\beta\kappa_0c_0}{\lambda} \int_0^t I_s(z)e^{\frac{\lambda z}{2}} dz \\ &\leq C^*(I_s^0, s(\cdot), \lambda, c_0) + \frac{2\beta\kappa_0c_0}{\lambda} \int_0^t I_s(z)e^{\frac{\lambda z}{2}} dz, \quad t > \epsilon_0 \end{aligned} \tag{3.7}$$

from $I_s(0) \geq 0$ and (3.3). Finally the Gronwall inequality yields

$$0 \leq I_s(t) \leq C^* e^{-\left(\frac{\lambda}{2} - \frac{2\beta\kappa_0c_0}{\lambda}\right)t}, \quad t > \epsilon_0.$$

Thus, for $(\beta, \kappa_1(t), \kappa_2(t), \kappa_3(t))$ satisfying $0 < \beta\kappa_0 < \min\{\frac{\lambda^2}{4c_0}, 1\}$, we have $\lim_{t \rightarrow \infty} I_s(t) = 0$. Then $\lim_{t \rightarrow \infty} c(t) = \lim_{t \rightarrow \infty} d(t) = 0$ follows immediately. The proof is complete. \square

Remark 3.2. This result ensures that, if the external input cases are limited in the sense of (3.3) and $\beta\kappa_0 \in (0, 1)$ is small enough, all the infected patients will eventually disappear in terms of our dynamical system. It is easy to understand the smallness of $\beta\kappa_0$, that is, low infection ratio, or high isolation ratio, or satisfactory performance of both vaccination ratio and vaccines. Since the patient’s infection ratio β is not clear at present stage, the efficient actions of increasing $\kappa_i(t)$ for $i = 1, 2, 3$ are crucial to the elimination of disease finally.

Now we verify our model in terms of the epidemic data in Yangzhou from July 28 to August 23, 2021 numerically. From news report, we know the daily number of the confirmed patients $j(t)$ and then the cumulative number $J(t)$ in the interval $[0, t]$. In our simulations, we take the parameters in our model as

$$I_s^0 = 554, \beta = 0.864, \kappa_1 = 0.998, \kappa_2 = 0.5, \kappa_3 = 0.92, p_1 = 0.9, p_2 = 0.6,$$

$$\tau_j = 9.8, \tau_{c_1} = 12, \tau_{c_2} = 17, \tau_{d_1} = 22, \tau_{d_2} = 30$$

and $\lambda = 0.55$ in the Gamma functions, then compute the numerical values $j_t(t)$ and $J_t(t)$ from our model, which simulate the practical data $j_e(t), J_e(t)$ in Yangzhou. The results are given in Table 1, while the behavior of real data $(j_e(t), J_e(t))$ and the simulant data $(j_t(t), J_t(t))$ are shown in Fig. 1.

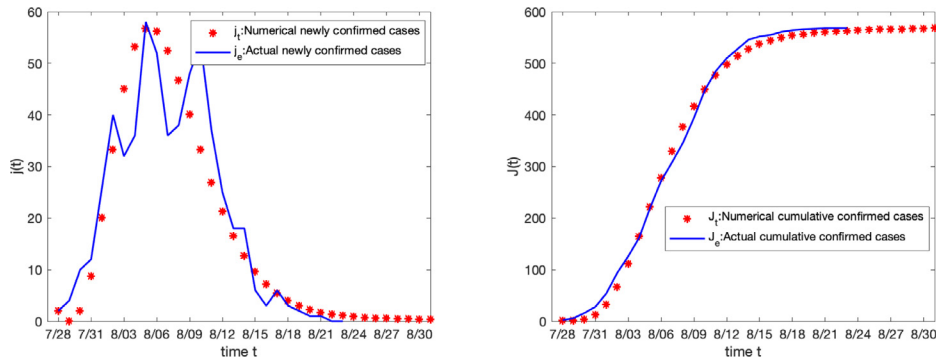


Fig. 1. The simulation behavior with Gamma distribution for Yangzhou epidemic data: daily confirmed patients (j_e, j_t) (left) and cumulative confirmed patients (J_e, J_t) (right).

These numerical results verify the validity of the proposed model rigorously, if we have appropriate initial value I_s^0 . Moreover, it is also found in our simulation process that the numerical performances are not sensitive for the parameters in the distribution functions, which shows the robustness of our proposed model. However, it is still an open problem for getting suitable values of I_s^0 and κ_i for $i = 1, 2, 3$. Mathematically, these values can be identified from extensive measurements by solving some inverse problems, which should be further studied in the future.

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