A mathematical model reveals the influence of NPIs and vaccination on SARS-CoV-2 Omicron Variant

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Abstract An SVEIR SARS-CoV-2 Omicron variant model is proposed to provide some insights to coordinate non-pharmaceutical interventions (NPIs) and vaccination. Mathematically, we define the basic reproduction number $R_0$ and the effective reproduction number $R_e$ to measure the infection potential of Omicron variant and formulate an optimal disease control strategy. Our inversion results imply that the sick period of Omicron variant in the United States is longer than that of Delta variant in India. The decrease in the infectious period of the infection with infectiousness implies that the risk of hospitalization is reduced; but the increasing period of the infection with non-infectiousness signifies that Omicron variant lengthens the period of nucleic acid test being negative. Optimistically, Omicron’s death rate is only a quarter of Delta’s. Moreover, we forecast that the cumulative cases will exceed 100 million in the United States on February 28, 2022, and the daily confirmed cases will reach a peak on February 2, 2022. The results of parameters sensitivity analysis imply that NPIs are helpful to reduce the number of confirmed cases. In particular, NPIs are indispensable even if all the people were vaccinated when the efficiency of vaccine is relatively low. By simulating the relationships of the effective reproduction number $R_e$, the vaccination rate and the efficacy of vaccine, we find that it is impossible to achieve the herd immunity without NPIs while the efficiency of vaccine is lower than 88.7%. Therefore, the herd immunity area is defined by the evolution of relationships between the vaccination rate and the efficacy of vaccine. Finally, we present that the disease-induced mortality rate demonstrates the periodic oscillation and an almost periodic function is deduced to match the curve. A discussion completes the paper.

Keywords SVEIR Omicron model · Reproduction numbers · NPIs · Vaccines · Sensitivity analysis · Herd immunity

Mathematics Subject Classification 34A34 · 34D20 · 92D30

1 Introduction

Coronavirus disease 2019 (COVID-19), caused by a novel virus of the coronavirus genus (SARS-CoV-2), has lasted for more than two years and caused a Once-in-a-Century global crisis [49]. Despite scientists worldwide racing to develop antiviral drugs and vaccinations against COVID-19 having been recently distributed around the world, the global economy is experiencing the worst plunge in recent history amid fears of further deterioration of the COVID-19 situation [13]. Since the outbreak of COVID-19 was first detected in December 2019 in Wuhan, China [29], many authors have studied the transmission dynam-
ics of COVID-19 by various means and methods [3,4,8,12,19,37,43,54–56].

As same as all viruses, SARS-CoV-2 which causes COVID-19 pandemic changes over time. For those changes, most of them do not impact on the virus’s properties. However, there may be some changes that have an important impact on the characteristics of the virus, such as how easily it is to spread, the severity of the associated diseases, the performance of vaccines and therapeutic medicines, the applications of the diagnostic tools, and/or the other public health and control measures [50]. For example, the B.1.1.529 (Omicron) variant, which was first identified in South Africa in early November 2021 [7], has rapidly become the dominant variant in many countries. The published research on live-virus neutralization assays revealed that Omicron could be escape antibody neutralization by the BNT162b2 messenger RNA vaccine (PfizerioNTech) [35].

Generally, population-wide rapid nucleic acid testing, isolation, sterilizing, and social distancing, which are called NPIs (non-pharmaceutical interventions), have played an important role to prevent and control the transmission of SARS-CoV-2 [15]. For example, many countries have been enforcing NPIs, such as social distancing (also called contact restrictions) and travel restrictions, to control the development of the SARS-CoV-2 [23,24]. Tian et al. [44] showed that the confirmed COVID-19 cases outside Wuhan would have decreased to 744,000 (± 156,000) due to the Wuhan travel ban or the national emergency response. Pavelka et al. [32] have investigated the influence of population-wide rapid antigen testing on SARS-CoV-2 prevalence in Slovakia. Their results showed that the prevalence decrease was not solely contributed by infection control measures, while the addition measures, such as the isolation and quarantine of household members of those testing positivity, were also required. Since SARS-CoV-2 is transmitted by droplets and aerosols, Cheng et al. [6] showed that the surgical masks were effective on preventing virus spread under conditions of low virus abundance (virus-limited). However, more advanced masks and other protective equipments were required in potentially virus-rich indoor environments, including medical centers and hospitals. Senapati et al. [39] revealed that it is necessary to take a higher intervention effort to control the disease outbreak within a shorter period of time in India. Further researches which has been proposed for considering the impact of NPIs on the spread of SARS-CoV-2 could be found in [27,33,34,42,45,51,53].

In fact, an excessive NPIs has restricted the development of the global economies and impacted on the general quality of life (in particular, mental health) [30]. That’s a general public perception that vaccines are the most effective defense to control the disease completely. Saad-Roy et al. [38] explored three scenarios of selection and found that a one-dose policy may increase the potential for antigenic evolution under certain conditions of partial population immunity. Moreover, they highlighted the critical need to test viral loads and quantify immune responses after one vaccine dose, and to ramp up vaccination efforts throughout the world. In consideration of limited initial supply of SARS-CoV-2 vaccine, Bubar et al. [4] used a mathematical model to compare five age-stratified prioritization strategies. Following some of the WHO-SAGE recommendations, Acuña-Zegarraa et al. [1] formulated an optimal control problem with mixed constraints to describe vaccination schedules.

Since someone was diagnosed with the Omicron variant of COVID-19 despite having received two shots of the vaccine, NPIs remain very indispensable to terminate the pandemic of SARS-CoV-2. Hence, it is necessary to identify strategies for safely relaxing nonpharmaceutical measures [18,31]. Drawing support from optimization-based control on an age-differentiated compartmental model, Grundel et al. [14] studied the relations of vaccination and social distancing. However, the published which coordinated NPIs and vaccination comprehensively to prevent the outbreak of Omicron variant is less common [1,4,38]. In this paper, we propose an SVEIR SARS-CoV-2 Omicron variant model to reveal the influence of NPIs and vaccination on SARS-CoV-2 Omicron variant in four key aspects. First, mathematically, we define the basic reproduction number $R_0$ and the effective reproduction number $R_e$ to measure the infection potential of Omicron variant and develop disease control strategies. Second, parameter inversion is conducted to explore the mechanism of Omicron variant and give some suggestions to stay home and isolate from other people. Third, sensitivity analysis finds the main factors affecting the spread of Omicron variant. Fourth, facing a low vaccination willingness and efficacy of vaccines, we explore the herd immunity area.

The remainder of this paper is structured as follows. We present the compartmental model in Sect. 2
A mathematical model reveals the influence of NPIs and describe the extinction and uniform persistence in Sect. 3. Section 4 is dedicated to the case study, and a discussion completes the paper in Sect. 5.

### 2 A compartmental model with NPIs and vaccination

In this paper, according to the propagation mechanism of SARS-CoV-2 [19, 25, 26, 36, 52], the individuals who have infected the virus are divided into the asymptomatic and symptomatic compartments. Furthermore, each of the asymptomatic and symptomatic class is divided into two groups, that is, one is contagious and other is non-contagious. Consider the transmission of Omicron variant with NPIs and vaccines, the population is divided into the following categories: Let $S$ be the susceptible individuals, $V$ be the vaccinated individuals, $E_1$ be the exposed individuals who are not contagious in the early stage, $E_2$ be the asymptomatic individuals who can infect the susceptible in the later stage, $I_1$ be the symptomatic individuals who are contagious in the early stages, $I_2$ be the exposed individuals who are not contagious in the later stage and $R$ be recovery individuals. $N$ denotes the total population, that is, $N = S + V + E_1 + E_2 + I_1 + I_2 + R$. Refer to [19], the population growth process can be described as in Fig. 1.

The model is given by an autonomous system of ordinary differential equations

\[
\begin{align*}
\frac{dS}{dt} &= \Lambda - \beta (1 - C_a) \frac{SE_2}{N} - \beta (1 - C_s) \frac{SI_1}{N} - \mu S - \theta R, \\
\frac{dV}{dt} &= \xi S - \beta (1 - C_a)(1 - \tau) \frac{VE_2}{N}, \\
\frac{dE_1}{dt} &= \beta (1 - C_a)(1 - \tau) \frac{VI_1}{N} - \mu V, \\
\frac{dE_2}{dt} &= \beta (1 - C_a) \frac{SE_2}{N} + \beta (1 - C_s) \frac{SI_1}{N} + \beta (1 - C_a)(1 - \tau) \frac{VE_2}{N} + \beta (1 - C_s)(1 - \tau) \frac{VI_1}{N} - \frac{E_1}{D_{E_1}} - \mu E_1, \\
\frac{dI_1}{dt} &= \frac{E_1}{D_{E_1}} - \frac{E_2}{D_{E_2}} - \mu E_2 - dE_2, \\
\frac{dI_2}{dt} &= \frac{E_2}{D_{E_2}} - \frac{I_1}{D_{I_1}} - \frac{I_2}{D_{I_2}} - \mu I_2, \\
\frac{dR}{dt} &= \frac{I_2}{D_{I_2}} - \mu R - \theta R,
\end{align*}
\]

(2.1)

where $\Lambda$ is the recruitment rate of susceptible class, $C_a$ and $C_s$ denote the intensity of NPIs for the individuals of the asymptomatic individuals and the symptomatic individuals who can infect the susceptible, respectively. $\beta$ denotes the effective contact rate, $\xi$ denotes the vaccination coverage rate, $\theta$ is the antibody disappear rate of recovery class, $\mu$ is the natural death rate of the population, $D_{E_1}$ and $D_{E_2}$ are the period of the lengths of the asymptomatic individuals who are not contagious in the early stages and the asymptomatic individuals who can infect the susceptible in the later stage, respectively. $0 \leq \tau \leq 1$ denotes the vaccine efficacy ($\tau = 1$ represents a vaccine that offers 100% protection against infection, $\tau = 0$ models a vaccine that offers no protection at all). $D_{I_1}$ and $D_{I_2}$ are the period of the lengths of the symptomatic individuals who are contagious in the early stages and the symptomatic individuals who can-
not infect the susceptible in the later stage, respectively. $d$ denotes the disease-induced mortality rate. Biologically, we could suppose that the number of total human population stabilizes at $N > 0$.

For simplicity, set $\psi_t(x^0)$ be the solution of (2.1) with initial value $\psi_0(x^0) = x^0 \in \mathbb{R}^T_+$, we have the following.

**Theorem 2.1** For any $x^0 \in \mathbb{R}^T_+$, system (2.1) has a unique nonnegative solution $\psi_t(x^0)$ with initial value $\psi_0(x^0) = x^0$, and all solutions are ultimately bounded and exist globally.

The proof of Theorem 2.1 can be seen in Appendix A.

**Remark 2.2** If we take $\tau = 0$ or $\xi = 0$ in (2.1), then the model consider that NPIs for the asymptomatic and symptomatic individuals who can infect the susceptible is the only measure. Similarly, let $C_a = C_s = 0$ in the above discussion, system (2.1) implies that the vaccination is only gotten involved. If $\tau = \xi = C_a = C_s = 0$, it means that there are no external factors involved (no vaccines, masks or other epidemic prevention measures).

## 3 Reproduction number

Reproduction numbers (ratio) are a crucial threshold parameter in the study of disease transmission. In epidemiology, the basic reproduction numbers is defined as the expected number of secondary cases produced by a single (typical) infection in a completely susceptible population and is used to measure the infection potential of an infectious disease [9, 46]. At the beginning of the transmission of coronavirus, based on likelihood and model analysis, Tang et al. [43] revealed that the basic reproduction number may be as high as 6.47, which showed that COVID-19 is highly infectious. By means of the basic reproduction number, Bubar et al. [4] found a highly mitigated spread during vaccine roll-out. Riley et al. [37] used a model of constant exponential growth and decay, and quantified this fall and rise in prevalence in terms of halving and doubling times and the basic reproduction number. Noting that an important quantity in epidemiological models, that is the reproduction number, Cuevas-Maraver et al. [8] investigated the role of lockdown measures in mitigating COVID-19 in Mexico using a comprehensive nonlinear ODE model. In this section, the definition and computation formulae of the basic reproduction number and the effective reproduction number for system (2.1) are established.

We first consider the disease-free solution of system (2.1). Let $E_1 = E_2 = I_1 = I_2 = R = 0$, then we have

$$\frac{dS}{dt} = \Lambda - \xi S - \mu S,$$

$$\frac{dV}{dt} = \xi S - \mu V. \tag{3.1}$$

By the similar arguments to those in [48], system (3.1) has a positive equilibrium $(S^*, V^*) = \left(\frac{\Lambda}{\tau + \tau}, \frac{\xi \Lambda}{\mu (\tau + \tau)}\right)$, which is globally attractive. Linearizing system (2.1) at the disease-free equilibrium $(S^*, V^*, 0, 0, 0, 0, 0)$, we get

$$\frac{dE_1}{dt} = \beta (1 - C_a) \frac{S^* E_2}{N} \left(1 - \tau \right) \frac{V^* I_1}{N} + \beta (1 - C_s) \frac{S^* I_1}{N} + \frac{E_1}{D_{E_1}} - \mu E_1,$$

$$\frac{dE_2}{dt} = E_1 \frac{E_2}{D_{E_1}} - \frac{E_2}{D_{E_2}} - \mu E_2 - d E_2,$$

$$\frac{dI_1}{dt} = \frac{E_2}{D_{E_2}} - I_1 \frac{I_1}{D_{I_1}} - \mu I_1 - d I_1. \tag{3.2}$$

Let

$$Y = \left( \begin{array}{ccc} 0 & F_1 & F_2 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{array} \right), Z = \left( \begin{array}{ccc} V_1 & 0 & 0 \\ -V_4 & V_2 & 0 \\ 0 & -V_5 & V_3 \end{array} \right),$$

where $F_1 = \beta (1 - C_a) \frac{S^* + (1 - \tau) V^*}{N}$, $F_2 = \beta (1 - C_s) \frac{S^* + (1 - \tau) V^*}{N}$, $V_1 = \frac{1}{D_{E_1}} + \mu$, $V_2 = \frac{1}{D_{E_2}} + \mu + d$, $V_3 = \frac{1}{D_{I_1}} + \mu + d$, $V_4 = \frac{1}{D_{E_1}}$ and $V_5 = \frac{1}{D_{E_2}}$. Then, we can rewrite (3.2) as

$$\frac{du}{dt} = (Y - Z)u. \tag{3.3}$$

Motivated by the concept of next generation matrices introduced in [9, 46], we define the effective reproduction number of system (2.1) as

$$\mathcal{R}_e := \rho(Y Z^{-1}), \tag{3.4}$$

where $\rho(A)$ denotes the spectral radius of a matrix $A$. By a simple calculation, the effective reproduction number of system (2.1) can be denoted that
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\[
R_e = \frac{\beta \Lambda (\mu + \xi (1 - \tau))(D_{E_2}(d + \mu + \frac{1}{\Lambda_1})(1 - C_a) + 1 - C_s)}{\mu N D_{E_1} D_{E_2} (\xi + \mu)(\mu + \frac{1}{\Lambda_1})(d + \mu + \frac{1}{\Lambda_2})}. \tag{3.5}
\]

The following two theorems give a threshold-type result on the extinction and uniform persistence of the disease in terms of \(R_e\) in the natural state. The proof of the following two theorems can be seen in Appendix A.

**Theorem 3.1** Assume \(R_e < 1\) holds, then the disease-free equilibrium \(E^* = (S^*, V^*, 0, 0, 0, 0, 0)\) of system (2.1) is globally attractive.

**Theorem 3.2** If \(R_e > 1\), then there exists \(\tilde{\varepsilon} > 0\) such that the solution \((S(t), V(t), E_1(t), E_2(t), I_1(t), I_2(t), R(t))\) of system (2.1) with initial data \(x^0\) in \(\mathbb{R}_+^7\) and \((E_1(0), E_2(0), I_1(0)) > \tilde{0}\) satisfies

\[
\liminf_{t \to \infty} E_1(t) > \tilde{\varepsilon}, \quad \liminf_{t \to \infty} E_2(t) > \tilde{\varepsilon}, \quad \liminf_{t \to \infty} I_1(t) > \tilde{\varepsilon}.
\]

**Remark 3.3** If \(\tau = 0\) or \(\xi = 0\) hold in the above discussion, then we call (3.4) as the NPIs reproduction number, denoted by \(R_e^N\). Similarly, in the case of \(C_a = C_s = 0\), (3.4) denotes the vaccines reproduction number \(R_e^V\). If \(\tau = \xi = C_a = C_s = 0\), then (3.4) is just the basic reproduction number \(R_0\). Furthermore, the conclusions of Theorem 3.1 and Theorem 3.2 remain available under different situations if we replace \(R_e\) by \(R_e^N, R_e^V\) and \(R_0\).

### 4 Numerical simulations

Omicron variant is a new type of SARS-CoV-2. In this section, we take the data in the United States to explore the transmission mechanism and predict the development trend of Omicron.

#### 4.1 Parameters inversions

We select the data in the United States showed in [21], from December 1, 2021, to January 30, 2022, and use the fminsearch function in MATLAB (R2019a 9.6.0.1072779) to perform parametric inversion. The value of the vaccination coverage rate \(\xi\) can be seen in [22]. In order to search the difference between Delta and Omicron variant, we compare the data in India [47] with that in the United States. The specific values are shown in Table 1.

According to Table 1, we find that the Omicron variant highly infectious in the sense that \(\beta = 0.8993\) and the sick period of Omicron variant in the United States is 0.91 days longer than that of Delta variant in India;

| Parameters | India | USA | Unit | Reference/Source |
|------------|-------|-----|------|------------------|
| \(\beta\)   | 0.70  | 0.8993 | –    | Calibrated       |
| \(D_{E_1}\) | 2.9   | 3.07 | Days | Calibrated       |
| \(D_{E_2}\) | 2.3   | 1.51 | Days | Calibrated       |
| \(D_{I_1}\) | 2.9   | 1.43 | Days | Calibrated       |
| \(D_{I_2}\) | 12    | 15.00 | Days | Calibrated       |
| \(d\)       | \(1.3 \times 10^{-4}\) | \(2.97 \times 10^{-5}\) | Day\(^{-1}\) | [21]          |
| \(C_a\)     | 0.20  | 0.1716 | –    | Calibrated       |
| \(C_s\)     | 0.20  | 0.2991 | –    | Calibrated       |
| \(\mu\)     | \(4 \times 10^{-5}\) | \(2.44 \times 10^{-5}\) | Day\(^{-1}\) | [20]          |
| \(N\)       | 1380004000 | 120552473 | People | [20]        |
| \(\Lambda\) | 65786 | 9877 | People/Day | [20]          |
| \(\theta\)  | 0     | 0.0085 | –    | Calibrated       |
| \(\tau\)    | 0.85  | 0.3300 | –    | [22]          |
| \(\xi\)     | 0.05  | 0.60  | –    | [22]          |
The decreasing of $D_I$ implies that the risk of hospitalization is reduced; but the increasing period of $D_I$ signifies that Omicron variant lengthens the period of nucleic acid test being negative; Optimistically, Omicron’s death rate is only a quarter of Delta’s. According to the value of $\theta$, we call for the four months interval of booster shot. Furthermore, if an asymptomatic person is confirmed or suspected of being infected with the Omicron variant, regardless of the result of parameters inversion, he should stay at home and isolate from other people for at least 6 days. The isolation time of symptomatic patients should be 3 days. Testing may be used to help determine when to end the isolation period [28].

4.2 Infection potential estimation

It then follows from the inversion results in above section that $R_0 = 8.883$, $R_e^N = 6.811$, $R_e^V = 5.952$ and $R_e = 4.564$. Therefore, it is shown that the infection potential of Omicron variant is very high without NPIs and vaccination in the United States. Even if one of NPIs and vaccination is implemented, Omicron variant might infect a large number of individuals. Obviously, the vaccination is better than that of NPIs for reducing the infectious potential of Omicron variant. Two countermeasures being implemented simultaneously remain the best way to protect oneself from COVID-19 and reduce its impact on our communities. Furthermore, we analysis the correlations between parameters and the effective reproduction number $R_e$ by random sample method with N=8000, the result is shown in Fig. 2. We can find that $R_e$ mainly ranges $[3, 5]$. This result reflects that the United States is facing a strong pressure for the prevention and control of Omicron.

4.3 Model fitting and trend predicting

In the following, we forecast the spreading trend of Omicron pandemic in the United States. By a calculation, the model fitting and prediction are shown in Fig. 3.

The discrete points and the blue line represent the real data of cumulative cases and daily increase cases from December 1, 2021, to January 30, 2022, in the United States, respectively. The red line and green line represent the simulation of cumulative cases and daily increase cases, respectively. We found that the dynamical results fit well with the statistical data [21] (see Fig. 3a). During December 1, 2021, to April 20, 2022, Fig. 3b forecasts that daily confirmed cases will maintain an upward trend and reach the maximum value 979360 on February 2, 2022. And the cumulative cases will be more than 100 million on February 28, 2022. Since the period of the symptomatic with infectiousness decreases, we think many people are treated at home. It leads to the fact that the number of reports is lower than the predicted. There is a huge problem that some of American without any face coverings and unwilling to take the vaccine. Furthermore, if the existing protection intensity and vaccine injection schedule are maintained, the numerical simulation forecasts COVID-19 in the United States will uniformly exist and form an endemic disease. Thus, it is imminent to tighten NPIs and accelerate vaccine programmes.

4.4 Sensitivity analysis

In this part, we compute Partial Rank Correlation Coefficients (PRCC) to identify the key factors which affect the change of $R_e$ and the total infectious cases. In our experiment, we set that the parameters have a significant effect when $p$ value < 0.01. The results can be seen in Fig. 4.

From Fig. 4a, we can easily see that the parameters $\beta, C_a, C_s, D_{E_2}, D_I$, and $\tau$ have significant effect on $R_e$. Furthermore, the effective contact rate $\beta$ and $R_e$ are positively correlated, which means that the increasing of effective contact rate can augment $R_e$. The parameters $C_a$ and $C_s$ are negatively correlated with $R_e$, which shows that NPIs are strengthened for the asymptomatic and symptomatic individuals with infectiousness can help to reduce the infection potential of Omicron variant. The parameters $D_{E_2}, D_I$, and $R_e$ are positively correlated, which reflect the peculiarity of Omicron. It is worth emphasizing that the vaccine efficacy $\tau$ plays an important role to control the transmission of COVID-19.

Figure 4b reveals that $\beta, C_a, C_s, \theta, D_{E_1}, D_{E_2}, D_I, \xi$ and $\tau$ effect mainly on the cumulative infected cases. Logically, the decrease in the effective contact rate $\beta$ can decrease the cumulative infected cases, which tell us that an environmental decontamination is an important measures to control the pandemic. For $C_a$ and $C_s$, we can strengthen NPIs, such as keeping social
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Fig. 2  The peculiarity of $R_e$

Fig. 3  a The model fitting of cumulative cases. b The model prediction of daily increase cases

distance, wearing a well-fitting mask, staying home and isolating from other people for asymptomatic and symptomatic individuals with infectiousness to control Omicron pandemic. Considering $\xi$, $\tau$ and $\theta$, we suggest that everyone should get vaccinated and boosted as soon as they are eligible, including people who have already infected COVID-19. Since $D_{E_1}$ denotes the period of the lengths of the asymptomatic individuals who are not contagious in the early stages, it’s easy to understand that the cumulative infected cases and $D_{E_1}$ are positively correlated. Since $D_{E_2}$ and $D_{I_1}$ reflect the peculiarity of Omicron, a drug intervention may be the most effective means.

The specific impact of parameters on the effective reproduction number $R_e$ and the cumulative infected individuals is shown in Table 2.

4.5 The influence of NPIs and vaccines

In order to visually reflect the effect of parameters, we increase the negative correlation parameters $C_a$, $C_s$,
Table 2 PRCC of the cumulative infected individuals ($C$) and the effective reproduction number ($R_e$)

| Parameter | $R_e$ PRCC | $p$ value | $C$ PRCC | $p$ value |
|-----------|------------|-----------|-----------|-----------|
| $\beta$   | 0.6773     | 0.0000    | 0.6888    | 0.0000    |
| $C_a$     | 0.8100     | 0.0000    | 0.8306    | 0.0000    |
| $C_s$     | -0.9360    | 0.0000    | -0.9294   | 0.0000    |
| $\theta$  | -0.0031    | 0.7829    | 0.0055    | 0.6243    |
| $D_{E_1}$ | -0.0161    | 0.1489    | -0.2503   | 0.0000    |
| $D_{E_2}$ | 0.9815     | 0.0000    | 0.9695    | 0.0000    |
| $D_{I_1}$ | 0.7344     | 0.0000    | 0.6613    | 0.0000    |
| $D_{I_2}$ | 0.0188     | 0.0931    | 0.0186    | 0.0960    |
| $\tau$    | -0.8061    | 0.0000    | -0.7994   | 0.0000    |
| $\xi$     | -0.0073    | 0.5124    | -0.0577   | 0.0000    |

The parameters $\xi$, $\tau$ to see the change of the cumulative infected (see Fig. 5).

Figure 5 reflects the trend of the impact of key and changeable parameters on cumulative infected individuals. It then follows from Fig. 5a and b that the cumulative infected individuals decrease with the increase in $C_a$ and $C_s$, and the decreasing range is relatively large. Figure 5c and d reveals that vaccination remains the best way to protect yourself from COVID-19 and reduce its impact on our communities. Obviously, the efficacy of vaccines has a significant effect on reducing the cumulative infected individuals. Due to the B.1.1.529 (Omicron) variant reducing the efficacy of vaccines and the low vaccination rate it will take months/years until herd immunity is achieved, NPIs play an important role in the prevention and control of COVID-19 pandemic. In the following, we accurately analyze the impact of Non-pharmaceutical interventions (NPIs) in the controlling of Omicron.

Figure 6 shows that $C_s$ and $C_a$ can effectively reduce the cumulative cases. Let $P$ be the decrement of numbers of infectious with the increase in $C_s$ and $C_a$ and other parameters remain unchanged. More intuitively, we have listed the specific numbers under the different intensity of NPIs (see Table 3).

It then follows from the data in Table 3 if $C_s$ is raised from 0.30 to 0.50 and $C_a$ is raised from 0.17 to 0.35, then there will be 19,808,021 fewer infected individual. Supposing that $C_a$ changes from 0.17 to 0.50 and $C_s$ is raised from 0.30 to 0.35, then there will...
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Fig. 5 The influence of $C_a$, $C_s$, $\xi$, $\tau$ on the cumulative infected individuals

Fig. 6 The decrement of numbers of infectious individuals with the increase in $C_s$ and $C_a$
be 20,079,078 fewer infected individual. If both $C_s$ and $C_a$ are increased to 0.50, then there will be 21,935,110 fewer infected individual. According to the disease-induced mortality rate in the United States, if we take $C_s = 0.35$, $C_a = 0.50$, then 35,185 people are saved; if $C_s = 0.50$, $C_a = 0.35$, then 34,710 people are saved; if $C_s = 0.50$ and $C_a = 0.50$, then 38,437 people are saved.

### 4.6 The relationship among $R_e$, NPIs and vaccine

At present, the efficacy of vaccine is not relatively low, and someone was diagnosed with the Omicron variant of COVID-19 despite having received two shots of the vaccine. In the following, under the assumption that all people are vaccinated Janssen COVID-19 Vaccine, we consider the relationships between $R_e$, $C_s$ and $C_a$ (see Fig. 7).

It can be seen from the above discussion that NPIs play a very significant role for the disease control. Figure 8 is the projection of Fig. 7 on the $C_s \times C_a$ plane. The green line in Fig. 8 represents $R_e = 1$. Figure 7 shows if $(C_a, C_s)$ belongs the area above the green line, then $R_e < 1$, while $R_e > 1$ in the area below the green line. Our numerical results shows that NPIs are indispensable even if all the people were vaccinated when the efficiency of vaccine is relatively low. In other words, in order to control the spread of Omicron, NPIs must be strengthened to make $C_s$ and $C_a$ in the area above the green line even if each people is vaccinated. Particularly, we suggest that NPIs should be strengthened, not weakened in the United States.

In the following, we study the role of the vaccine in the absence of NPIs.

### 4.7 Herd immunity

In the following, we look for the possibility of herd immunity in the United States. It is easy see that $\tau \xi$ indicates the proportion of antibody produced after vaccination. Let $C_a = C_s = \xi = \tau = 0$ and $R_0 = 8.883$. Theoretically, we conclude if $1 - \frac{1}{R_0} < \tau \xi$ is satisfied [10], then the herd immunity is formed.

From Fig. 10, it reveals that $\xi = 0.887$ when $\tau = 1$, and $\tau = 0.887$ if $\xi = 1$. The intersection of the area $C$ and $D$ is called the herd immunity line which satisfies $\tau \xi = 0.887$ and $C$ is the herd immunity area where the condition $\tau \xi > 0.887$ is satisfied. Since there is a huge problem that some of American without any face coverings and unwilling to take the vaccine, and the efficacy of vaccines is low against Omicron, everyone should get vaccinated and boosted as soon as they are eligible, including people who have already infected COVID-19.

### 4.8 Almost periodicity of disease-induced mortality rate

In the process of parameters analysis, we find that the disease-induced mortality rate demonstrates the periodic oscillation. To further confirm this, we use an
Fig. 7 When $\tau = 0.33$ and $\xi = 1$, the image in three dimensions of relationship among $R_{e}$, $C_{t}$ and $C_{a}$.

Fig. 8 When $\tau = 0.33$ and $\xi = 1$, the relationship between $C_{t}$ and $C_{a}$.

Fig. 9 When $C_{t} = 0$ and $C_{a} = 0$, the relationship between $\xi$ and $\tau$ and $R_{e}$. 

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Fig. 10 When \( C_a = 0 \) and \( C_e = 0 \), the relationship between herd immunity and \( \tau \) and \( \xi \)

Fig. 11 The simulation of the disease-induced mortality

almost periodic function

\[
d(t) = 2.687608 \times 10^{-5} + 1.419898 \times 10^{-5} \\
\times \cos((407/400) \sqrt{5} \pi t) + 1.317339 \times 10^{-6} \\
\times \sin((103/100) \sqrt{6} \pi t)
\]

to simulate the curve by MATLAB, and the simulation result can be shown in Fig. 11. It’s obviously that the surveillance and simulation fit very well. Since seasonal variations in temperature, rainfall, resource availability, contact rates, the birth and death rates of populations and immune defenses are ubiquitous and can exert strong pressures on population dynamics [2], we think that this result is reasonable.

5 Discussion

In this paper, according to the propagation mechanism of SARS-CoV-2 Omicron variant, an SVEIR model with non-pharmaceutical interventions (NPIs) and vaccination is proposed. By means of the basic reproduction number \( R_0 \) and the effective reproduction number \( R_e \), the theories of uniform persistence and extinction are also established. Our inversion results which come from the data of the United States imply that the duration of illness of Omicron variant is longer than that of Delta variant. Omicron variant admits a stronger infectious ability while the duration of infection is shorter. The decrease in the infectious period of the infection with infectiousness implies that the risk of hospitalization is reduced; but the increasing period of the infection with non-infectiousness signifies that Omicron variant lengthens the period of nucleic acid test being negative. Optimistically, Omicron’s death rate is only a quarter of Delta’s. Our results predict a persistent risk of the disease in the United States. Sensitivity analysis implies that non-pharmaceutical interventions (NPIs) and the efficacy of vaccine are the main factor to control the spread of disease. If the outbreak occurs repeatedly, we suggest that NPIs should be strengthened. Furthermore, it is shown that NPIs are indispensable even if all the people were vaccinated when the efficiency of vaccine is relatively low. In order to obtain the herd immunity, we speculate in numerical simulation that the minimum efficacy of vaccine is 88.7%. In the face of the crisis of confidence of vaccine and logistical challenges, the herd immunity area is given. Certainly, we expect that COVID-19 will die out as soon as possible by the efforts of people of all over the world.

Author contributions BW contributed to conceptualization and project administration. ZW and YW contributed to supervision. YX and JZ contributed to formal analysis. YX, JZ, ZW and ZM are involved in software. YX, JZ and BW were involved in writing—original draft. BW, ZW, YW, YX and JZ were involved in writing—review and editing. ZW and ZM contributed to data curation.

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Data availability The dataset used and/or analyzed during the current study is available from the published works.

Declarations

Conflict of interest The authors declare no conflict of interest

Ethical approval and consent to participate The study protocol was approved by School of Mathematics and Statistics of Lanzhou University.
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Appendix A: Proof of Theorems 2.1, 3.1 and 3.2

Proof of Theorem 2.1 Since system (2.1) is local Lip- schitz in $\mathbb{R}_+^2$, it follows from [11,17] that system (2.1) has a unique solution $\psi_t(x^0)$ with $\psi_0(x^0) = x^0$ on its maximal interval $[0, \sigma_v)$. Moreover, [40, Theorem 5.2.1] implies that $\psi_t(x^0) \geq 0$ for all $t \geq 0$ on its maximal interval.

Next, we show that the solution of system (2.1) remains bounded. Let

$$N(t) = S(t) + V(t) + E_1(t) + E_2(t) + I_1(t) + I_2(t) + R(t).$$

Then, we have

$$\frac{dN(t)}{dt} = \Lambda - \mu N(t) - d(E_2(t) + I_1(t)) \leq \Lambda - \mu N(t).$$

It follows from the comparison theorem that $N(t)$ is bounded with

$$\limsup_{t \to \infty} N(t) \leq \frac{\Lambda}{\mu}.$$ 

Hence, the solution of system (2.1) is ultimately bounded and [11,17] implies that $\sigma_v = \infty$.

Proof of Theorem 3.1 It is easy to see that $S(t)$ and $V(t)$ satisfy

$$\frac{dS}{dt} \leq \Lambda - \xi S - \mu S + \theta R$$

and

$$\frac{dV}{dt} \leq \xi S - \mu V,$$

respectively. Since system (3.1) has a unique positive constant solution $(S^*, V^*)$, which is global asymptotically stable, by the comparison theorem, for any $x^0 \in \mathbb{R}_+^2$ and $\epsilon > 0$, there exists $t_0 > 0$ such that

$$S(t) \leq S^* + \epsilon, V(t) \leq V^* + \epsilon, \quad \forall t \geq t_0.$$ 

It then follows that

$$\frac{dE_1}{dt} \leq \beta(1 - C_a) \frac{(S^* + \epsilon)E_2}{N} + \beta(1 - C_s) \frac{(S^* + \epsilon)I_1}{N} + \beta(1 - C_a)(1 - \tau) \frac{(V^* + \epsilon)E_2}{N} + \beta(1 - C_s)(1 - \tau) \frac{(V^* + \epsilon)I_1}{N} - \frac{E_1}{D_{E_1}} - \mu E_1,$$

$$\frac{dE_2}{dt} \leq \frac{E_1}{D_{E_1}} - \frac{E_2}{D_{E_2}} - \mu E_2,$$

$$\frac{dI_1}{dt} \leq - \frac{I_1}{D_{I_1}} - \mu I_1.$$ 

we consider the following system

$$\frac{dE_1}{dt} = \beta(1 - C_a) \frac{(S^* + \epsilon)E_2}{N} + \beta(1 - C_s) \frac{(S^* + \epsilon)I_1}{N} + \beta(1 - C_a)(1 - \tau) \frac{(V^* + \epsilon)E_2}{N} + \beta(1 - C_s)(1 - \tau) \frac{(V^* + \epsilon)I_1}{N} - \frac{E_1}{D_{E_1}} - \mu E_1, \quad (1)$$

$$\frac{dE_2}{dt} = \frac{E_1}{D_{E_1}} - \frac{E_2}{D_{E_2}} - \mu E_2,$$

$$\frac{dI_1}{dt} = \frac{E_2}{D_{E_2}} - \frac{I_1}{D_{I_1}} - \mu I_1.$$ 

Since $\bar{R}_e < 1$, then $\omega(-Z + Y) < 0$, where $\omega(-Z + Y)$ is the exponential growth bound [48]. By the continuity of spectral bound, there exists a sufficiently small $\epsilon_1 > 0$ such that $\omega(-Z + Y^\ast) < 0$ for $0 < \epsilon < \epsilon_1$, which implies that the trivial solution of system (1) is globally asymptotically stable. By the comparison theorem of ordinary differential equation, we deduce that $E_1 \to 0, E_2 \to 0, I_1 \to 0$ as $t \to \infty$. It then follows that system (3.1) is the limiting system of $S, V$ equation in system (2.1). We also could get that $I_2, R$ equation admits the limiting system

$$\frac{dI_2}{dt} = - \frac{I_2}{D_{I_2}} - \mu I_2,$$

$$\frac{dR}{dt} = - \frac{I_2}{D_{I_2}} - \mu R - \theta R. \quad (2)$$

It is easy to see that the solutions in (2) convergence to $(0, 0)$. Finally, by the theory of asymptotically autonomous systems (see, e.g., [5]), we conclude that the solution of system (2.1) converges to $(S^*, V^*, 0, 0, 0, 0, 0)$. This confirms the global attractivity of $\mathcal{E}^*$ for system (2.1) under the condition $\bar{R}_e < 1$, and hence, we complete the proof.

Proof of Theorem 3.2 Define

$$X = \mathbb{R}_+^7,$$

$$X_0 := \{(S, V, E_1, E_2, I_1, I_2, R) \in X : E_1 > 0, E_2 > 0, I_1 > 0\}$$

$$\partial X_0 := X \setminus X_0$$

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Then, $X_0$ and $\partial X_0$ are relatively open and closed in $\mathbb{R}^7$, respectively. For any $x^0 \in X_0$, let $\psi_t(x^0)$ be the unique solution of system (2.1) with initial data $x^0$. It is easy to see that $X_0$ is a positively invariant set. According to the arguments in Section 2, the solution of (2.1) is ultimately bounded in $X$, which implies that $\psi_t : X \to X$ is point dissipative on $X$. It follows from [16, Theorem 3.4.8] that $\psi_t$ has a global compact attractor $\mathcal{A}$.

Define

$$M_\beta := \{ x^0 \in \partial X_0 : \psi_t(x^0) \in \partial X_0, \forall t \geq 0 \}$$

and

$$\mathcal{M} := \{ x^0 \in X : x^0 = S^*, x^0 = V^*, x^0 = x^*_0 = 0 \}.$$  

We now show that $M_\beta = \mathcal{M}.$

For any $x^0 \in \mathcal{M}$, the solution $\psi_t(x^0)$ satisfies $E_1(t, x^0) = 0$, $E_2(t, x^0) = 0$, $I_1(t, x^0) = 0$ for all $t \geq 0$. Hence, $x^0 \in M_\beta$ and $\mathcal{M} \subset M_\beta$.

For any $x^0 \in \partial X_0 \setminus \mathcal{M}$, there is $(x^0_0, x^0_1, x^0_2)$ such that $(x^0_0, x^0_1, x^0_2) = (E_1(0), E_2(0), I_1(0)) > (0, 0, 0)$.

**Case 1** Let $E_1(0) > 0$. Then, the third equation of system (2.1) satisfies

$$\frac{dE_1}{dt} \geq -\frac{E_1}{DE_1} - \mu E_1.$$  

Furthermore, we deduce that $E_1(t) > 0$ for all $t \geq 0$.

From the fourth and fifth equation of system (2.1), it is easy to get that $E_2(t) > 0$ and $I_1(t) > 0$ for all $t \geq 0$.

**Case 2** Let $E_2(0) > 0$. By a similar arguments to those in **Case 1**, we can obtain that $(E_1(t), E_2(t), I_1(t)) \gg (0, 0, 0)$ for all $t \geq 0$.

**Case 3** Let $I_1(0) > 0$. By a similar arguments to those in **Case 1**, we can obtain that $(E_1(t), E_2(t), I_1(t)) \gg (0, 0, 0)$ for all $t > 0$.

Then, $M_\beta \subset \mathcal{M}$. Hence, $M_\beta = \mathcal{M}$.

We claim that $W^s(\mathcal{M}) \cap X_0 = \emptyset$, where $W^s(\mathcal{M})$ is the stable manifold of $\mathcal{M}$. Let $\bar{\lambda} = \beta(1 - C_a) \frac{(N^* - 2\epsilon - \tau V^*)}{N}$, $\bar{\eta} = \beta(1 - C_a) \frac{(N^* - 2\epsilon - \tau V^*)}{N}$.

Denote

$$\bar{\lambda} = \bar{\lambda}, \quad \bar{\eta} = \bar{\eta}.$$  

Let

$$\tilde{\lambda} = \begin{pmatrix} F_1^\epsilon & F_2^\epsilon \\ 0 & 0 \\ 0 & 0 \end{pmatrix},$$  

$$\tilde{\eta} = \begin{pmatrix} 0 & F_1^\epsilon \\ 0 & 0 \\ 0 & 0 \end{pmatrix}.$$  

Since $\mathcal{R}_e > 1$, then $\omega(Y - Z) > 0$. By the continuity of spectral bound, there exists a sufficiently small $\epsilon_1 > 0$ such that $\omega(\tilde{Y}_e - Z) > 0$ for $0 < \epsilon \leq \epsilon_1$.

**Claim:** If $x^0 \in X_0$, then

$$\limsup_{t \to \infty} d(\psi_t(x^0), \mathcal{M}) \geq \epsilon_1.$$  

On the contrary, we assume that there exists $\tilde{x}^0 \in X_0$ such that $\limsup_{t \to \infty} d(\psi_t(\tilde{x}^0), \mathcal{M}) < \epsilon_1$. It then follows that there exists $t_0 > 0$ such that

$$S^* - \epsilon_1 < S(t) < S^* + \epsilon_1, V^* - \epsilon_1 < V(t) < V^* + \epsilon_1$$

for all $t \geq t_0$. Hence, we have

$$\frac{dE_1}{dt} \geq \beta(1 - C_a)(S^* - \epsilon_1)E_2 - \beta(1 - C_a)(1 - \tau)(V^* - \epsilon_1)I_1 - \frac{E_1}{DE_1} - \mu E_1,$$

$$\frac{dE_2}{dt} \geq \frac{E_1}{DE_1} - \frac{E_2}{DE_2} - \mu E_2,$$

$$\frac{dI_1}{dt} \geq \frac{E_2}{D_{I1}} - \frac{I_1}{D_{I1}} - \mu I_1.$$  

Since $-Z + \tilde{Y}_e$ is irreducible and essentially nonnegative, it has a positive eigenvector associated with $\omega(-Z + \tilde{Y}_e) > 0$. By the comparison theorem of ordinary differential equations, we have $\lim_{t \to \infty} E_1(t) = \infty$, $\lim_{t \to \infty} E_2(t) = \infty$, $\lim_{t \to \infty} I_1(t) = \infty$, a contradiction. The claim is proved.

The set $M_\beta = \mathcal{M}$ is an isolated invariant set and acyclic. By [41, Theorem 4.6], we conclude that system (2.1) is uniformly persistent in $X_0$ whenever $\mathcal{R}_e > 1$. That is, there is a $\tilde{\epsilon} > 0$ such that

$$\liminf_{t \to \infty} E_1(t) > \tilde{\epsilon}, \liminf_{t \to \infty} E_2(t) > \tilde{\epsilon}, \liminf_{t \to \infty} I_1(t) > \tilde{\epsilon}.$$  

This completes the proof.

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