Theoretical Analysis of the SIRVVD Model for Insights Into the Target Rate of COVID-19/SARS-CoV-2 Vaccination in Japan

YUTO OMAE\textsuperscript{1,} (Member, IEEE), MAKOTO SASAKI\textsuperscript{1,} JUN TOYOTANI\textsuperscript{1,}
KAZUYUKI HARA\textsuperscript{1,} (Member, IEEE), AND HIROTAKA TAKAHASHI\textsuperscript{2}

\textsuperscript{1}College of Industrial Technology, Nihon University, Chiba 275-8575, Japan
\textsuperscript{2}Research Center for Space Science, Advanced Research Laboratories, Tokyo City University, Tokyo 158-0082, Japan

Corresponding author: Yuto Omae (oomae.yuto@nihon-u.ac.jp)

The work of Yuto Omae was supported in part by the Telecommunications Advancement Foundation under Grant 20203002. The work of Jun Toyotani was supported by the Japan Society for the Promotion of Science (JSPS) Grant-in-Aid for Scientific Research (C) under Grant 21K04535.

ABSTRACT The effectiveness of the first dose of vaccination for COVID-19 is different from that of the second dose; therefore, in several studies, various mathematical models that can represent the states of the first and second vaccination doses have been developed. Using the results of these studies and considering the effects of the first and second vaccination doses, we can simulate the spread of infectious diseases. The susceptible-infected-recovered-vaccination1-vaccination2-death (SIRVVD) model is one of the proposed mathematical models; however, it has not been sufficiently theoretically analyzed. Therefore, we obtained an analytical expression for the number of infected persons by considering the numbers of susceptible and vaccinated persons as parameters. We used the solution to determine the target vaccination rate for decreasing the infection numbers of the COVID-19 Delta variant (B.1.617) in Japan. Furthermore, we investigated the target vaccination rates for cases with strong or weak variants by comparing with the COVID-19 Delta variant (B.1.617). This study contributes to the mathematical development of the SIRVVD model and provides insights into the target vaccination rate for decreasing the number of infections.

INDEX TERMS SIRVVD model, vaccination, herd immunity, COVID-19, SARS-CoV-2.

I. INTRODUCTION

As of December 2021, the COVID-19 pandemic continues to be a global concern. To overcome the spread of COVID-19, various countermeasures have been implemented, such as telework \[1\], lockdown \[2\], restrictions on movement \[3\], airport quarantine \[4\], tracing apps \[5\], and regular disinfection of public spaces \[3\]. These countermeasures are known as “non-pharmaceutical interventions (NPIs)”. NPIs are considered to reduce the number of infected persons. Nonetheless, many people oppose the concept of lockdown and restrictions on movement \[3\]. During the COVID-19 crisis and the execution of various NPIs, several COVID-19 vaccines, including BNT162b (Pfizer) \[6\], mRNA-1273 (Moderna) \[7\], and ChAdOx1 (AstraZeneca) \[8\], were developed. There are some NPIs that can be lifted by vaccination \[9\].

Therefore, increasing the number of vaccinated persons and gaining insight into the effects of vaccination are important. Mathematical simulation is a desirable approach to gain insight into the effect of vaccination on the spread of infection. In several studies, COVID-19 country-based simulations (Malaysia \[10\], Saudi Arabia \[11\], Spain \[12\], United States \[13\], Pakistan \[14\], and others) have already been reported. The fast simulation method of differential equations is a susceptible-infected-recovered-vaccination (SIRV) model \[15\]–\[19\]. However, these models can represent only the first vaccination dose. For COVID-19, the effects of the first and second doses on prevention of infection are different; for the COVID-19 Delta variant (B.1.617), the effects of the first and second doses on the prevention of infection are 35.6% and 88.0%, respectively \[20\].

As a result, mathematical models that represent the first and second doses of vaccination have been proposed \[21\]–\[25\]. Using these studies, we can investigate the effect of vaccination on the number of infected persons.
However, insights into the solutions of differential equations including the vaccination effects are insufficient. Therefore, it is difficult to represent the condition to stop or spread the virus infection in the form of a mathematical function. Against the background, we aim to find an analytical expression of the infectious number, including the coefficient related to vaccination based on the SIRVVD model [21] that is one of the differential equations for representing the first and second doses of vaccination.

Currently, many studies exist on the analysis of medicals or biosciences based on various advanced mathematical models, including the respiratory mechanics model [26] and the fractional model of the blood ethanol concentration system [27]. Differential equation–based studies for investigating the spread of the COVID-19 infection are also same, and there are many theoretical studies, e.g., [28]–[30]. Because these studies provide important insights, the objective of our study is also important.

To stop the spread of infection, it is important to achieve herd immunity through vaccination. Some previous studies have reported various target values for achieving herd immunity through vaccination [31]–[35]. Because the target vaccination rate to achieve herd immunity depends on the lifestyle of the country (wearing masks and social distancing) [36], it is important to obtain target values of vaccination for each country. Therefore, we calculated the target rate of vaccinated persons in the scenario of the spread of the COVID-19 Delta variant (B.1.617) in Japan based on the solution of infectious numbers of the SIRVVD model. In addition, we investigated target rates in the case of becoming strong or weak variants compared with the COVID-19 Delta variant (B.1.617).

II. SIRVVD MODEL

A. DIFFERENTIAL EQUATIONS

In [21], the SIRVVD model was applied to reproduce the spread of infection in actual Japan using real data. Because our study aimed to theoretically analyze the model, the structure of differential equations will be comprehensively described herein. The SIRVVD model [21] for representing the first and second doses of vaccination is defined as:

$$\dot{S}(t) = -\beta(t)S(t)I(t) + \theta_1 V_1(t) + \theta_2 V_2(t) - \alpha_1 S(t) - \theta_0 R(t),$$

(1)

$$\dot{V}_1(t) = \alpha_1 S(t) - \theta_1 V_1(t) - \alpha_2 V_1(t) - (1 - \sigma_1)\beta(t) V_1(t) I(t),$$

(2)

$$\dot{V}_2(t) = \alpha_2 V_1(t) - (1 - \sigma_2)\beta(t) V_2(t) I(t) - \theta_2 V_2(t),$$

(3)

$$\dot{I}_0(t) = \beta(t)S(t)I(t) - \gamma I_0(t),$$

(4)

$$\dot{I}_1(t) = (1 - \sigma_1)\beta(t) V_1(t) I(t) - \gamma I_1(t),$$

(5)

$$\dot{I}_2(t) = (1 - \sigma_2)\beta(t) V_2(t) I(t) - \gamma I_2(t),$$

(6)

$$\dot{R}(t) = \gamma \sum_{i\in\{0,1,2\}} (1 - \delta_i) I_i(t) - \theta_0 R(t),$$

(7)

$$\dot{D}(t) = \gamma \sum_{i\in\{0,1,2\}} \delta_i I_i(t),$$

(8)

where the dot notation represents the time derivative, i.e., \(f'(t) := \frac{df(t)}{dt}\). Further, \(S(t)\) represents the number of susceptible persons; \(I_{j\in\{0,1,2\}}(t)\) represents the number of infected persons of \(j\) times the dose of vaccination \((j = 0\) implies no vaccination). \(V_{j\in\{1,2\}}(t)\) represents the number of vaccinated persons of \(j\) times the dose of vaccination. \(R(t)\) represents the number of recovered persons, and \(D(t)\) represents the number of dead persons. Moreover, the total infection number \(I(t)\) is defined as

$$I(t) = I_0(t) + I_1(t) + I_2(t).$$

(9)

Next, we describe the coefficient parameters as follows: \(\beta(t)\) represents infectivity; \(\theta_{j\in\{0,1,2\}}\) represents the average antibody period as \(j\) times the dose of vaccination \((j = 0\) implies natural infection); \(\sigma_{j\in\{1,2\}}\) represents the transition rate to \(V_j(t)\); \(\delta_{j\in\{0,1,2\}}\) represents the effectiveness of \(j\).
times the dose of vaccination (decreasing the probability of infection); \( \gamma^{-1} \) denotes the average infection period; and \( \delta_{j} \in (0,1] \) denotes the fatality rate of the persons of \( j \) times the dose of vaccination.

The state transitions are shown in Figure 1. Paths 1, 7, and 8 indicate that new infected persons are produced by multiplying the infectivity \( \beta(t) \), persons with the possibility of infection (i.e., \( S(t) \), \( V_1(t) \), and \( V_2(t) \)), and infected persons \( I(t) \). This indicates that when these values are high, many new infected persons are produced. Moreover, the infection probability and fatality rate of vaccinated persons \( V_j(t) \) decrease due to vaccination effects \( \sigma_{j} \in [1,2] \) (paths 7 and 8) and \( \delta_{j} \in [1,2] \) (path 10).

Then, the total population \( N \) is
\[
N = S(t) + I(t) + V_1(t) + V_2(t) + R(t) + D(t), \quad \forall t \in R^+_0, \tag{10}
\]
where \( R^+_0 \) represents the set of plus real numbers that include zero. To maintain a constant total population, \( N \) includes \( D(t) \), which is the number of deaths due to an infectious disease. The model does not consider deaths by other means, except an infectious disease.

**B. ANALYTICAL EXPRESSION OF THE INFECTION NUMBER**

It is desirable that the function of the infectious number \( I_{j}(t) \) is clear to investigate the infectious spread. However, it is difficult to obtain an analytical solution in the absence of assumptions because \( I_{j}(t) \) is complex (including various functions). Therefore, we assume that \( S(t) \), \( V_1(t) \), \( V_2(t) \), and \( \beta(t) \) are not time-dependent functions but the constants of \( S \), \( V_1 \), \( V_2 \), and \( \beta \), respectively. Then, the total number of persons, \( N' \), with the possibility of infection is given by
\[
N' = S + V_1 + V_2. \tag{11}
\]

\( N \) includes persons with no possibility of contracting infection (e.g., dead persons); however, \( N' \) does not include these. Note that if \( I(t) \), \( R(t) \), and \( D(t) \) are sufficiently small, \( N \approx N' \). \( I_{j}(t) \) defined by Equations (4)-(6) can be simplified as
\[
\begin{align*}
\dot{I}_0(t) &= (\beta_0 - \gamma)I_0(t) + \beta_0 I_1(t) + \beta_0 I_2(t) \\
\dot{I}_1(t) &= \beta_1 I_0(t) + (\beta_1 - \gamma)I_1(t) + \beta_1 I_2(t) \\
\dot{I}_2(t) &= \beta_2 I_0(t) + \beta_2 I_1(t) + (\beta_2 - \gamma)I_2(t),
\end{align*}
\tag{12}
\]
where
\[
\beta_0 = \beta S, \quad \beta_1 = \beta (1 - \sigma_1) V_1, \quad \beta_2 = \beta (1 - \sigma_2) V_2. \tag{13}
\]

Here, if we define
\[
\begin{align*}
I(t) &= (I_0(t) \ I_1(t) \ I_2(t))^T, \\
A &= \begin{pmatrix}
\beta_0 - \gamma & \beta_0 & \beta_0 \\
\beta_1 & \beta_1 - \gamma & \beta_1 \\
\beta_2 & \beta_2 & \beta_2 - \gamma
\end{pmatrix}, \tag{14}
\end{align*}
\]
then Equation (12) can be simplified into
\[
\dot{I}(t) = AI(t) \tag{15}
\]
as first-order separable simultaneous differential equations. Here, we transform it into
\[
\int \frac{1}{I(t)} dI(t) = A \int dt, \tag{16}
\]
after which we can represent
\[
\begin{align*}
I(t) &= e^{At}I(0) \\
&= e^{\mu_1 \mu_2 \mu_3}I(0) \\
&= Pe^{U}P^{-1}I(0), \tag{17}
\end{align*}
\]
where \( U \) represents the diagonal matrix of \( A \). These relationships can be represented by
\[
P^{-1}AP = U = \begin{pmatrix}
\lambda_0 & 0 & 0 \\
0 & \lambda_1 & 0 \\
0 & 0 & \lambda_2
\end{pmatrix}, \tag{18}
\]
where \( P \) comprises the eigenvectors of the coefficient matrix \( A \), and \( \lambda_{j} \in (0,1] \) represents the eigenvalues. In other words, the solution \( I(t) \) is clear by calculating the eigenvectors and eigenvalues of the coefficient matrix \( A \).

Let a \( 3 \times 3 \) unit matrix be denoted by \( E \); then,
\[
\det(A - \lambda E) = (\beta_0 - \gamma - \lambda)(\beta_1 - \gamma - \lambda)(\beta_2 - \gamma - \lambda) + 2\beta_0 \beta_1 \beta_2 - \beta_0 \beta_2 (\beta_1 - \gamma - \lambda) - \beta_1 \beta_2 (\beta_0 - \gamma - \lambda) - \beta_0 \beta_1 (\beta_2 - \gamma - \lambda) = (\gamma + \lambda)^2 (\beta_0 + \beta_1 + \beta_2 - \gamma - \lambda). \tag{19}
\]
In addition, the eigenvalues \( \lambda \) are obtained by solving the eigenvalue \( \det(A - \lambda E) = 0 \) as
\[
\begin{align*}
\lambda_{j} \in (0,1] &= -\gamma, \tag{20} \\
\lambda_2 &= \beta_0 + \beta_1 + \beta_2 - \gamma. \tag{21}
\end{align*}
\]
From the eigenvalues, \( P \) comprises eigenvectors that can be represented as
\[
P = \begin{pmatrix}
-1 & -1 & \beta_0 \\
0 & 1 & \beta_1 \\
1 & 0 & \beta_2
\end{pmatrix}, \tag{22}
\]
where
\[
B = \beta_0 + \beta_1 + \beta_2, \quad \beta \neq 0. \tag{23}
\]
The solution \( I(t) \) of the differential equation \( \dot{I}(t) \) can be represented by substituting Equations (20)-(22) into Equation (17). Then, solutions \( I_{j}(t) \) are represented as
\[
I_{j}(t) = X_{j} e^{\left[\beta (s+\sum_{i \in [1,2]}(1-\sigma_{i})V_{i})-\gamma \right] t} + Y_{j} e^{-\gamma t}. \tag{24}
\]
The coefficients are given by

\[ X_0 = \frac{SI(0)}{S + \sum_{i \in \{1,2\}} (1 - \sigma_i)V_i}, \]

\[ X_1 = \frac{(1 - \sigma_i)V_i}{S + \sum_{i \in \{1,2\}} (1 - \sigma_i)V_i}, \]

\[ Y_0 = \frac{\sum_{i \in \{1,2\}}[(1 - \sigma_i)V_iI(0) - SI(0)]}{S + \sum_{i \in \{1,2\}} (1 - \sigma_i)V_i}, \]

\[ Y_i = \frac{S + (1 - \sigma_k)V_iI(0) - (1 - \sigma_i)V_iI(0) + I_k(0))}{(S + \sum_{i \in \{1,2\}} (1 - \sigma_i)V_i)}, \]

where,

\[ l \in L = \{1, 2\}, \]

\[ k \in L \setminus \{l\}, \]

\[ S + \sum_{i \in \{1,2\}} (1 - \sigma_i)V_i \neq 0. \] (26)

These equations require constant values of \( S(t), V_1(t), \) and \( V_2(t); \) thus, they are not suitable for long-term simulations.

**C. TARGET VACCINATION RATE**

\( \gamma^{-1} \) represents the average infection period; therefore, \( \gamma > 0. \) This indicates that \( e^{-\gamma t}, \) the second term of Equation (24), becomes zero depending on time \( t. \) The dynamics \( e^{-\gamma t} \) of some average infection periods \( (\gamma^{-1} = 5, 10, 15, \) and \( 20 \) days) are shown in Figure 2. This indicates that \( e^{-\gamma t} \) becomes zero in approximately a month. Therefore, we assume

\[ e^{-\gamma t} \sim 0. \] (27)

Furthermore, because the coefficients of Equation (24) are constants, each solution \( I_{j \in \{0,1,2\}}(t) \) can be approximated by

\[ I_{j \in \{0,1,2\}} \sim e^{\left(\beta \left( S + \sum_{i \in \{1,2\}} (1 - \sigma_i)V_i \right) - \gamma \right) t}, \]

\[ e^{\left(\beta \left( N' - \sum_{i \in \{1,2\}} \sigma_iV_i \right) - \gamma \right) t}. \] (28)

\( S \) was deleted by Equation (11). Therefore, we obtain the following theorems:

\[ \beta(N' - \sum_{i \in \{1,2\}} \sigma_iV_i) - \gamma > 0 \Rightarrow I_j(t) \text{ increases,} \] (29)

\[ \beta(N' - \sum_{i \in \{1,2\}} \sigma_iV_i) - \gamma = 0 \Rightarrow I_j(t) \text{ does not change,} \] (30)

\[ \beta(N' - \sum_{i \in \{1,2\}} \sigma_iV_i) - \gamma < 0 \Rightarrow I_j(t) \text{ decreases,} \] (31)

where, \( j \in \{0, 1, 2\}. \) The spread of infection stops when a scenario as indicated in Equation (30) is created. We collected terms on virus parameters to the right side and vaccination parameters on the left side to clarify this scenario. As a result, Equation (30) can be simplified as

\[ N' - \sigma_1V_1 - \sigma_2V_2 = \gamma \beta^{-1}. \] (32)

Here, \( N' \) denotes the total number of persons with a probability of infection; \( \sigma_{j \in \{1,2\}} \) represents the effectivity of vaccination; and \( V_{j \in \{1,2\}} \) represents the number of vaccinated persons. Thus, the left side represents the total number of people who have the probability of infection considering the vaccination (the left side is small when using a highly effective vaccine). The longer the infection period and the higher the infectivity, the smaller the right term because \( \gamma^{-1} \) represents the average infection period and \( \beta \) represents the infectivity. In addition, in the case a virus has a long infection period and high infectivity, a highly effective vaccine is required to stop the spread of infection.

Here, let \( V_{2 \text{obj}} \) denote the target number of the second vaccination dose required to stop the spread of infection. We can obtain \( V_{2 \text{obj}} \) by solving Equation (30) for \( V_2 \) as

\[ V_{2 \text{obj}} = \frac{N' - \sigma_1V_1 - \gamma \beta^{-1}}{\sigma_2}. \] (33)

Furthermore, the target rate of the second vaccination dose required to stop the infection spread \( P_{2 \text{obj}} \) is

\[ P_{2 \text{obj}} = \frac{V_{2 \text{obj}}}{N'}. \] (34)

Because the unit of \( P_{2 \text{obj}} \) is a rate, when it is multiplied by 100, the unit becomes a percentage.

**D. LIMITATIONS OF VACCINATION**

We consider the condition of not stopping the infection spread even if all persons receive the second vaccination dose. We obtain the following theorem because this scenario would mean substituting \( V_2 = N', \) \( V_1 = 0 \) for Equation (29).

\[ \sigma_2 < 1 - \frac{\gamma}{\beta N'} \Rightarrow I_{j \in \{0,1,2\}}(t) \text{ increases when } V_1 = 0 \text{ and } V_2 = N'. \] (35)

Even if all persons receive the second vaccination dose, the infection number continues to increase when the effectivity, \( \sigma_2, \) of the vaccination second dose is insufficient. In contrast, the condition of Equation (35) is not satisfied when the
vaccination has perfect prevention (i.e., $\sigma_2 = 1$) because $N' > 0$, $\beta > 0$, and $\gamma > 0$. In other words, the spread of infection stops when developing a vaccination with a perfect prevention effect (i.e., $\sigma_2 = 1$) and administering a vaccination to all persons (i.e., $V_2 = N'$). However, it is important to know the condition of Equation (35) because achieving this is difficult.

E. CONDITIONS UNDER WHICH THE VACCINE IS NOT REQUIRED

We consider conditions under which the vaccine is not required; this implies substituting $V_2 = 0$, $V_1 = 0$ for Equation (31). We can obtain a simplified equation as

$$0 > 1 - \frac{V}{\beta N'} \Rightarrow \text{when } V_{j \in \{1,2\}} = 0, I_{j \in \{0,1,2\}} \text{ decreases.}$$

(36)

F. VALIDITY OF THE TARGET NUMBER $V_2^{\text{OBJ}}$.

Here, we check whether the infection spread stops by satisfying $V_2^{\text{OBJ}}$. Therefore, we conduct the SIRVVD model–based simulations (i.e., Equations (1) – (8)) assuming a simple scenario. The fixed parameters are

- Simulation period $t_{\text{end}} = 10$ [days]
- Total population $N = 2 \times 10^6$
- Initial infection numbers of $j$ times vaccinated persons $I_{j \in \{0,1,2\}}(0) = 10^3$
- Initial dead persons $D(0) = 0$
- Initial recovered persons $R(0) = 0$
- Initial susceptible persons $S(0) = N - \sum_j I_{j}(0) - D(0) - R(0) - \sum_j V_j(0)$
- Effectivity of the first dose of vaccination $\sigma_1 = 0.50$
- Effectivity of the second dose of vaccination $\sigma_2 = 0.90$
- Vaccination rate $\alpha_{j \in \{1,2\}} = 0$

The parameters validated by multiple values are

- Removal rate $\gamma \in \{\gamma_{\text{base}}, 2 \times \gamma_{\text{base}}\}$ ($\gamma_{\text{base}} = 0.05$)
- Infectivity $\beta \in \{\beta_{\text{base}}, 2 \times \beta_{\text{base}}\}$ ($\beta_{\text{base}} = 10^{-7}$)
- Initial number of vaccinated persons (first dose) $V_1 \in \{0, N/2\}$

and we adopted $V_2 \in \{0 \times V_2^{\text{obj}}, 0.2 \times V_2^{\text{obj}}, \cdots, 2.0 \times V_2^{\text{obj}}\}$ as the number of vaccinated persons to verify the target value $V_2^{\text{OBJ}}$.

The simulation results for cases $V_1 = 0$ and $V_1 = N/2$ are shown in Figures 3 and 4, respectively. The horizontal axis represents $V_2$, while the vertical axis represents $I(t_{\text{end}})/I(0)$. In addition, the black dashed line represents $I(t_{\text{end}})/I(0) = 1$, while the red dashed line represents $V_2 = V_2^{\text{OBJ}}$. $I(t_{\text{end}})/I(0) > 1$ indicates that the infection number increases, whereas $I(t_{\text{end}})/I(0) < 1$ implies that it decreases. In all cases, when $V_2 = V_2^{\text{OBJ}}$, $I(t_{\text{end}})/I(0) = 1$. Further, the infection number increases ($I(t_{\text{end}})/I(0) > 1$) when the number of vaccinated persons is low ($V_2 < V_2^{\text{OBJ}}$). In contrast, the infection number decreases ($I(t_{\text{end}})/I(0) < 1$) when the number of vaccinated persons is sufficient ($V_2 > V_2^{\text{OBJ}}$). Therefore, the target number of the second dose of vaccination, $V_2^{\text{OBJ}}$ based on Equation (33) can be considered reliable.

III. TARGET RATE $P_2^{\text{OBJ}}$ UNDER THE SCENARIO OF THE COVID-19 DELTA VARIANT (B.1.617) IN JAPAN

We calculate the target rate $P_2^{\text{OBJ}}$ of the second dose of vaccination assuming the case of the COVID-19 Delta variant (B.1.617) in Japan.

A. INFECTIVITY OF THE COVID-19 DELTA VARIANT (B.1.617) IN JAPAN

Let $\beta_{\text{delta}}$ denote the infectivity of the COVID-19 Delta variant (B.1.617) in Japan. According to previous research [37], infectivity $\beta$ can be represented as

$$\beta = r \gamma S^{-1},$$

(37)
where \( r \) denotes the effective reproduction number. Further, \( \beta \) calculated using Equation (37) includes the effects of various countermeasures since the effective reproduction number includes these effects. We can thus obtain infectivity \( \beta_{\delta} \) if we know the effective reproduction number of the delta variant (B.1.617) in Japan. According to the COVID-19 advisory board for the Japanese Government [38], after August 2021, all infected people had the delta variant (B.1.617). Thus, the effective reproduction number during this period can be attributed to the COVID-19 Delta variant (B.1.617). However, on August 31, 2021, the rates of people who received the first and second vaccination doses were 11% and 46%, respectively (total rate: 57%) [39]. Therefore, we consider that the infectivity of the Delta variant (B.1.617) is underestimated due to the effect of vaccination when considering the newer cases of infection during this period.

Thus, we estimated the infectivity of the Delta variant (B.1.617) in Japan using data from the period when vaccination had not started (before February 2021). According to the COVID-19 advisory board for the Japanese Government [40], the Delta variant (period: August 2021) had an infection rate 1.9 times higher than that of the regular COVID-19 virus (period: December 2020) in Japan. Moreover, because no people were vaccinated in December 2020, we consider the infectivity of the Delta variant (B.1.617) as 1.9 times that of this period. We assume

\[
\beta_{\delta} = 1.9 \beta_{\text{regular}},
\]

(38)

where \( \beta_{\delta} \) and \( \beta_{\text{regular}} \) represent the infectivities of the Delta variant (B.1.617) and regular COVID-19 virus, respectively.

The maximum value of the effective reproduction number in December 2020 in Japan was \( r = 1.18 \) [41]. Kobayashi et al. estimated 0.13 to 0.17 as the \( \gamma \) parameter of COVID-19 in Japan [42]. We adopted \( \gamma = 0.13 \) because we consider optimism to be undesirable, and this leads to a long infection period. The total population in Japan is approximately \( S = 1.2 \times 10^8 \) persons. We obtain \( \beta_{\text{regular}} \approx 1.28 \times 10^{-5} \) as the infectivity of the COVID-19 regular virus by substituting these parameters in Equation (37). Moreover, the infectivity of the Delta variant (B.1.617) is \( \beta_{\delta} \) as \( 2.43 \times 10^{-9} \) by substituting \( \beta_{\text{regular}} \) for Equation (38).

### B. Effectiveness of the BNT162B (Pfizer) Vaccine

In a previous study, Bernal et al. [20] reported the effect of the BNT162b (Pfizer) vaccine on reducing the probability of infection with the COVID-19 Delta variant (B.1.617). The effect of the first dose was 35.6% (22.7~46.4%), and that of the second dose was 88.0% (85.3~90.1%). Thus, we adopted \( \sigma_1 = 0.356 \) and \( \sigma_2 = 0.880 \).

### C. Target Number \( V_{2}^{\text{obj}} \) and Target Rate \( P_{2}^{\text{obj}} \) in Japan

We consider the target number \( V_{2}^{\text{obj}} \) and target rate \( P_{2}^{\text{obj}} \) in Japan for the spread of the Delta variant (B.1.617). We assume that all persons who received the first dose would receive the second dose, i.e., \( V_1 = 0 \). Further, the total population in Japan is approximately \( 1.2 \times 10^8 \). \( V_{2}^{\text{obj}} \) can be represented by substituting these values for Equation (33) as

\[
V_{2}^{\text{obj}} = \frac{N' - \sigma_1 V_1 - \gamma \beta^{-1}}{\sigma_2} = \frac{1.2 \times 10^8 - 0.356 \times 0 - 0.13 \times (2.43 \times 10^{-9})^{-1}}{0.880} \approx 7554 \times 10^4.
\]

(39)

Thus, the spread of infection will stop when 7554 \times 10^4 persons receive the second dose of vaccination. The target rate is \( P_{2}^{\text{obj}} = V_{2}^{\text{obj}} / N' = 0.6295 \). A vaccination rate of approximately 63% is required for the total population in Japan to stop the spread of infection. The target rate of 63% is for the case of the lifestyle of December 2020 in Japan. If we obtain the lifestyle values before the appearance of COVID-19 (before December 2019), a target rate of approximately 63% will be insufficient.

Shen et al. [43] reported that approximately 72.5% of the population in various countries have been administered the COVID-19 vaccine. We believe that the target vaccination rate can be achieved in Japan because the target rate \( P_{2}^{\text{obj}} = 0.63 \) is lower than the reported global value of 72.5%. In Japan, on December 27, 2021, 78% of the total population received the second dose of vaccination [39], and the infection number decreased (e.g., the average number of new cases of infection per day from November 1, 2021, to December 31, 2021, was lower than 3 persons per million persons [44]).

### D. Comparison with Other Studies

The target rates of vaccinated persons reported by other researchers are listed in Table 1. These are target rates for achieving herd immunity to stop the spread of infection. We examined the different values in this study.

One reason for the different values is that the target rate is dependent on the lifestyle of each country. For example, Shen et al. [36] reported that the target vaccination rate is dependent on wearing masks and social distancing.

The target rate of vaccinated persons to stop the spread of infection is not the same because the lifestyles of each country are not the same. Thus, it is necessary to calculate the target

| Paper            | Area        | Effect* | Target rate |
|------------------|-------------|---------|-------------|
| Agarwal et al. [31] | World       | 82.5%   | 45 - 60 %   |
| Gunzel et al. [32] | United States | 70%    | 80 %        |
| Gunzel et al. [32] | United States | 95%    | 60 %        |
| Kadokoda [33]    | -           | 100%    | 62 - 72 %   |
| Kadokoda [33]    | -           | 95%     | 63 - 76 %   |
| Fontanet et al. [34] | France   | -       | 67 %        |
| Liu et al. [35]  | China       | 90%     | 83 - 92 %   |
| Ours             | -           | 88%     | 63%         |
| **: Prevention effect of vaccine.** | **: Case of lifestyle in December 2020 in Japan.** |
Y. Omae et al.: Theoretical Analysis of SIRVVD Model for Insights Into Target Rate

**IV. CASES OF STRONG OR WEAK NEW VARIANTS**

In the near future, there is a possibility of new variants occurring; for example, the COVID-19 Omicron variant (B.1.1.529) [45]. Thus, we consider the cases of strong or weak new variant comparisons with the Delta variant (B.1.617). We consider the following conditions.

- Changing the average infection period $\gamma^{-1}$
- Changing infectivity $\beta$
- Changing vaccination effectivity $\sigma_2$

For simulation parameters, we considered the average infection periods ranging from 2–20 days, and the infectivity ranging from 0.2–3 times of $\beta_{\text{delta}}$. In addition, we considered three patterns as vaccination effectiveness: the case of perfect prevention ($\sigma_2 = 1$); that of BNT162b (Pfizer) effectivity ($\sigma_2 = 0.88$ [20]); and that of a lower effectivity compared with that of BNT162b (Pfizer) ($\sigma_2 = 0.70$). Thus, we adopted combinations of the following parameters

$$
\gamma^{-1} \in \{2, 4, \ldots, 20\}, \\
\beta \in \{0.2 \times \beta_{\text{delta}}, 0.4 \times \beta_{\text{delta}}, \ldots, 3.0 \times \beta_{\text{delta}}\}, \\
\sigma_2 \in \{1, 0.88, 0.70\}.
$$

Furthermore, we calculated the target rate $P_{\text{obj}}^2$ to stop the spread of infection.

The target rates $P_{\text{obj}}^2$ in the case of perfect prevention ($\sigma_2 = 1$) are shown in Figure 5. The vertical axis represents the average infection period $\gamma^{-1}$, and the horizontal axis represents the infectivity $\beta$ (1.0 $\times$ $\beta_{\text{delta}}$ represents the Delta variant infectivity). The percentage values are $P_{\text{obj}}^2$, and the white hatchings on the left lower side indicate the case of satisfying Equation (36), i.e., vaccination is not required to stop the spread of infection. This result suggests that the spread of infection will stop if almost all persons receive the second dose of vaccination even if the new variant has a higher infectivity than that of the Delta variant (B.1.617).

However, the perfect prevention vaccine is not realistic. Therefore, we calculate the case of $\sigma_2 = 0.88$, as shown in Figure 6. Painted black markers represent the results satisfying Equation (35), i.e., even if all persons receive the second dose of vaccination, the spread of infection does not stop. This scenario occurs in the cases of long infection periods and high infectivity. For such a scenario, we consider not only vaccinations but also lockdowns, such as stay-at-home orders.

Finally, the case of a new variant that leads to a weaker vaccination effect ($\sigma_2 = 0.70$) is shown in Figure 7. There are many cases wherein only vaccination cannot stop the spread of the infection caused by a new strong variant.
The results indicate that it is important to develop more high-effect vaccines to overcome the new strong variant.

V. LIMITATIONS OF THE STUDY

Because there are some limitations in this study, a careful interpretation must be made. Herein, we describe these points.

- We assumed the constants of $S$, $V_1$, and $V_2$. However, the vaccination effects disappeared by the elapsed time [45], [46]. For example, if the average antibody period $\theta_2^{-1}$ is excessively short, $V_2(t)$ decreases rapidly. Moreover, with the spread of infection, $S(t)$ decreases depending on the number of elapsed days. Therefore, when the average antibody period is long and the number of susceptible persons is high, the results shown in Figures 5-7 hold true.

- Herein, we discussed the target rate of only the second dose. Nonetheless, there is also the vaccination strategy of the delayed second dose for prioritizing the first dose vaccination for as many persons as possible. This strategy is evaluated in previous studies [23]–[25]. When the effect of the first dose on infection prevention $\sigma_1$ is significant, we consider this strategy to be effective. In a future study, we will investigate this effect, considering the country to be Japan.

- Regarding other studies on the simulation of the spread of COVID-19, there are mathematical models that consider the states of “asymptomatic” and “exposed” people (e.g., Moore et al. [47]). However, the mathematical model explained in Subsection II-A cannot represent these states. When considering asymptomatic and exposed people, the results shown in Figures 5-7 may change.

- In Subsection II-C, we assumed $e^{-\gamma t} \sim 0$. Regarding Figure 2, in the case where the average infection period is below 20 days, we assume that no major problems occur. However, when the average infection period is excessively long, the assumption of $e^{-\gamma t} \sim 0$ may be inappropriate. This indicates that there is a possibility that the second term of Equation (24) cannot be ignored.

- In Japan, there are several large cities (e.g., Tokyo, Osaka, etc.), and these are separated by distance. However, the classical SIR model assumes that the population is completely mixed [48]. Because the SIRVVD model also makes this assumption, the results of this study are macroscopic and approximate.

- The results are the assumption of the lifestyle in December 2020 in Japan. When the lifestyles of persons change, the target vaccination rate also changes. For example, with increasing activities of persons, the target vaccination rate should be increased.
Y. Omae et al.: Theoretical Analysis of SIRVVD Model for Insights Into Target Rate

FIGURE 7. Target rates $P_{2}^{\text{obj}}$ ($\sigma_2 = 0.70$), which are the vaccination rates to stop the spread of infection ($P_{2}^{\text{obj}}$ are multiplied by 100 and expressed as percentages). The horizontal axis represents the infectivity $\beta$ expressed by the infectivity of the Delta variant $\beta_{\text{delta}}$. The vertical axis represents the average infection period $\gamma^{-1}$. The white hatchings on the left lower side indicate the case of satisfying Equation (36). This indicates that vaccination is not required to stop the spread of infection. The painted black markers represent the results satisfying Equation (35). This demonstrates that even if all persons receive the second dose of vaccination, the infection number continues to increase.

number of vaccinated persons for achieving herd immunity also increases. If the lifestyles of persons considerably change, the results shown in Figures 5-7 may not be reliable.

- We adopted $\sigma_2 = 0.88$, which is the effect against the COVID-19 Delta variant [20]. However, in a previous study, the prevention effect of the Pfizer vaccine against the COVID-19 Omicron variant was reported to be 44% (i.e., $\sigma_2 = 0.44$) [46]; this value is very small compared with those observed in the cases of the Delta variant. Compared with the Delta variant, in the case of the Omicron variant, achieving herd immunity with vaccination is difficult. Therefore, the development of vaccines with improved effectiveness against various new variants is important.

VI. CONCLUSION

We found the analytical expressions of $I_{\in{0,1,2}}(t)$ of the differential equations $I_{\in{0,1,2}}(t)$ of the SIRVVD model. Furthermore, we proposed a method to determine $P_{2}^{\text{obj}}$, which is the target vaccination rate required to stop the spread of infection. Assuming the COVID-19 Delta variant (B.1.617) in Japan, we estimated $P_{2}^{\text{obj}}$ to be approximately 63%. Further, we calculated the target vaccination rate by assuming a new strong or weak variant (Figures 5, 6, and 7). We consider that these values are important to control the spread of infection.

However, we have not described which population groups should be vaccinated. Moreover, we assumed that the number of vaccines is sufficient. Marković et al. [49] investigated which groups (young, healthy, elderly, or other higher-risk groups) should be vaccinated and the appropriate vaccination strategy when vaccination availability is limited. Piraveenan et al. [50] described that when the spread of an infectious disease decreases, the perceived risk of people decreases. In this case, the number of people being vaccinated is low; therefore, an appropriate payoff for being administered the vaccine is required to stop the spread of infection. Against the background, they proposed a framework based on game theory. Moreover, the willingness to get vaccinated varies based on the country [43]. Therefore, our study as well as other previous studies are important when considering vaccination strategies.

REFERENCES

[1] G. Buomprisco, S. Ricci, R. Perri, and S. De Sio, “Health and telework: New challenges after COVID-19 pandemic,” Eur. J. Environ. Public Health, vol. 5, no. 2, Feb. 2021, Art. no. em0073, doi: 10.21601/ejeph/0073.
Y. Omoe et al.: Theoretical Analysis of SIRVD Model for Insights Into Target Rate

[2] H. Lau, V. Khosrawipour, P. Cobba, A. Mikolajczyk, J. Schubert, J. Bania, and T. Khosrawipour, “The positive impact of lockdown in Wuhan on containing the COVID-19 outbreak in China,” J. Travel Med., vol. 27, no. 3, pp. 1–7, Mar. 2020, doi: 10.1097/MTM.0000000000000376.

[3] T. Czyzponia et al., “The benefits, costs and feasibility of a low incidence COVID-19 strategy,” Lancet Regional Health-Eur., vol. 13, Feb. 2022, art. no. 100824, doi: 10.1016/j.lanepfo.2021.100294.

[4] T. Sekizuka, K. Itokawa, K. Yatsu, R. Tanaka, M. Hashino, T. Kawano-Sugaya, M. Ohnishi, T. Wakita, and M. Kuroda, “COVID-19 genome surveillance at international airport quarantine stations in Japan,” J. Travel Med., vol. 28, no. 2, Feb. 2021, art. no. taa217, doi: 10.1093/mtm/taa217.

[5] N. Ahmed, R. A. Michelin, W. Xue, S. Ruj, R. Malaney, S. S. Kanhere, A. B. Vogel et al., “The benefits, costs and feasibility of a low incidence COVID-19 strategy,” J. Travel Med., vol. 10, no. 4, pp. 717–731, Dec. 2019, doi: 10.26713/ctma.v10i4.1172.

[6] Y. Omae, Y. Kakimoto, M. Sasaki, J. Toyotani, K. Hara, Y. Gon, and H. Takahashi, “SIRVVD model-based verification of the effect of first and second doses of COVID-19/SARS-CoV-2 vaccination in Japan,” Math. Biosci. Eng., vol. 19, no. 1, pp. 1026–1040, Jan. 2022, doi: 10.3934/mbe.2022047.

[7] Y. Omae, Y. Kakimoto, M. Sasaki, J. Toyotani, K. Hara, Y. Gon, and H. Takahashi, “SIRVVD model-based verification of the effect of first and second doses of COVID-19/SARS-CoV-2 vaccination in Japan,” Math. Biosci. Eng., vol. 18, no. 5, pp. 6506–6526, Jul. 2021, doi: 10.3934/mbe.2021235.
[38] Advisory Board for Countermeasures in Japan Against COVID-19 (54th), Document 3–3 (p.120). Accessed: Dec. 24, 2021. [Online]. Available: https://www.mhlw.go.jp/content/109000000000840251.pdf

[39] Our World in Data, Statistics and Research: Coronavirus (COVID-19) Vaccinations. Accessed: Dec. 24, 2021. [Online]. Available: https://ourworldindata.org/covid-vaccinations

[40] Advisory Board for Countermeasures in Japan Against COVID-19 (40th), Document 3–3 (p.84). Accessed: Dec. 12, 2021. [Online]. Available: https://www.mhlw.go.jp/content/10900000000796736.pdf

[41] Coronavirus Disease (COVID-19) Situation Report in Japan, Accessed: Dec. 12, 2021. [Online]. Available: https://www.kyoeikai.or.jp

[42] G. Kohyashi, S. Sugasawa, H. Tamae, and T. Oza, “Predicting intervention effect for COVID-19 in Japan: State space modeling approach,” BioSci. Trends, vol. 14, no. 3, pp. 174–181, Jun. 2020, doi: 10.5582/bst.2020.03133.

[43] M. Snehota, J. Vlckova, K. Cizkova, J. Vachutka, H. Kolarova, et al., “Acceptance of a vaccine against COVID-19—A systematic review of surveys conducted worldwide,” Bratislava Med. J., vol. 122, no. 8, pp. 538–547, 2021, doi: 10.4149/bmj_2021_086.

[44] Our World in Data, Statistics and Research: Coronavirus (COVID-19) Cases. Accessed: Jan. 5, 2022. [Online]. Available: https://ourworldindata.org/covid-cases

[45] N. Andrews et al. (2021). Effectiveness of COVID-19 Vaccines Against the Omicron (B.1.1.529) Variant of Concern. [Online]. Available: https://www.medrxiv.org/content/10.1101.2021.12.14.21267615v1

[46] B. Reiner. COVID-19 Model Update: Omicron and Waning Immunity. Accessed: Mar. 20, 2022. [Online]. Available: https://www.healthdata.org/special-analysis/omicron-and-waning-immunity

[47] S. Moore, E. M. Hill, M. J. Tildesley, L. Dyson, and M. J. Keeling, “Vaccination and non-pharmaceutical interventions for COVID-19: A mathematical modelling study,” Lancet Infectious Diseases, vol. 21, no. 6, pp. 793–802, Jun. 2021, doi: 10.1016/S1473-3099(21)00143-2.

[48] S. Pyne, A. K. S. Vullikanti, and M. V. Marathe. “Big data applications in health sciences and epidemiology,” in Handbook of Statistics, vol. 33, Amsterdam, The Netherlands: Elsevier, 2015, pp. 171–202, doi: 10.1016/B978-0-444-63492-4.00008-3.

[49] R. Marković, M. Šterk, M. Marhl, M. Perc, and M. Gosiak, “Socio-demographic and health factors drive the epidemic progression and should guide vaccination strategies for best COVID-19 containment,” Results Phys., vol. 26, Jul. 2021, Art. no. 104433, doi: 10.1016/j.rinp.2021.104433.

[50] M. Piraveenan, S. Sawleshwarkar, M. Walsh, I. Zahlotska, S. Bhattacharyya, H. H. Farooqui, T. Bhattachar, A. Karian, M. Murthakar, S. Zodpey, K. S. M. Rao, P. Pattison, A. Zomaya, and M. Perc, “Optimal governance and implementation of vaccination programmes to contain the COVID-19 pandemic,” Roy. Soc. Open Sci., vol. 8, no. 6, Jun. 2021, Art. no. 210429, doi: 10.1098/rsos.210429.

MAKOTO SASAKI received the Ph.D. degree in science from Tokyo University, Japan, in 2009. He worked as an Assistant Professor at Kyushu University, from 2010 to 2020, and moved to Nihon University, in 2021. He was a Visiting Researcher at Aix-Marseille University, France, in 2018, and at Warwick University, U.K., in 2019. His research interests include statistical theory of turbulence, development of data-driven science methods, big-data analysis, and machine learning for understanding complex phenomena. He is a member of the Physical Society of Japan (JPS) and the Japan Society of Plasma Science and Nuclear Fusion (JSPF).

JUN TOYOTANI received the Ph.D. degree from the Graduate School of Industrial Engineering, Nihon University. He is a Professor with the Department of Industrial Engineering and Management, College of Industrial Technology, Nihon University. His research interests include data science, digital marketing analysis, and research on streaming operations using AI. He is a member of the Japan Society for Engineering Education, the Information Processing Society of Japan, and the Japan Society of Directors.

KAZUYUKI HARA (Member, IEEE) received the B.Eng. and M.Eng. degrees from Niigata University, in 1979 and 1981, respectively, and the Ph.D. degree from Kanazawa University, in 1997. He was involved in NEC Home Electronics Corporation, from 1981 to 1987. He joined as a Lecturer at the Toyama Polytechnic College, in 1987, and an Associate Professor at the Tokyo Metropolitan College of Technology, in 1998, and became a Professor, in 2005. He became a Professor at Nihon University, in 2010. His current research interest includes statistical mechanics of on-line learning. He is a Senior Member of the IPSJ and a member of the JPS, IEICE, and IEICE.

HIROTAKA TAKAHASHI received the Ph.D. degree in science from the Graduate School of Science and Technology, Niigata University, Japan, in 2005. He was a PD at the Max Planck Institute for Gravitational Physics (Albert Einstein Institute), Germany, from September 2005 to August 2007. He is a Faculty Staff Member with various universities. He has been a Professor with the Advanced Research Laboratories, Research Center for Space Science, and the Graduate School of Integrative Science and Engineering, Tokyo City University, Japan, since September 2020. His research interests include adaptive data analysis methods, information theory, and gravitational wave physics and astronomy (general relativity). He is a member of the Institute of Electrometrics, Information and Communication Engineers (IEICE) and the Physical Society of Japan (JPS).

YUTO OMAE (Member, IEEE) received the Ph.D. degree in engineering from the Department of Information Science and Control Engineering, Nagaoka University of Technology, Japan, in 2016. He was a Postdoctoral Researcher at the Japan Institute of Sports Sciences and an Assistant Professor at the Tokyo College, National Institute of Technology. He has been an Assistant Professor with the Department of Industrial Engineering and Management, College of Industrial Technology, Nihon University, Japan, since April 2019. His research interests include intelligent informatics, machine learning, and mathematical model. He is a member of the Institute of Electronics, Information and Communication Engineers (IEICE), the Japanese Society for Artificial Intelligence (JSAI), and the Japan Society for Fuzzy Theory and Intelligent Informatics (J-SOFT).