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IMPLEMENTATION OF THE SEQHRV3 EPIDEMIOLOGICAL MODEL FOR MITIGATING COVID-19 AND OTHER VACCINE-PREVENTABLE DISEASE OUTBREAKS IN INDONESIA

FARAH KRISTIANI¹, ROBYN IRAWAN^{1,*}, SALSA DIAH RETNO ARUMSARI¹, WINARDI EMMANUEL SETIAWAN², SANDRA CHRISTELLA GUNAWAN¹

¹Center for Mathematics and Society, Faculty of Science, Parahyangan Catholic University, Jl. Ciumbuleuit No. 94, Bandung 40141, Indonesia

²Center for Medical Science and Technology and Healthcare Equity, Faculty of Medicine, Parahyangan Catholic University, Jl. Ciumbuleuit No. 94, Bandung 40141, Indonesia

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Abstract. The global landscape faces continuous challenges from infectious diseases, often presenting unique and complex epidemic patterns due to emerging pathogens (e.g., COVID-19). This complexity underscores the necessity for effective control strategies. This study analyzes disease spread dynamics and develops an epidemiological model for assessing outbreak prevention strategies, focusing on vaccination as an intervention variable to model the dynamic progression of the disease more accurately, thereby simulating real-world epidemiological scenarios. The analysis employs the SEQHRV3 compartmental model using the most recent pandemic data (COVID-19), with parameters estimated from national health insurance, vaccination, and mortality databases. The system of differential equations was solved numerically over 5,000 days, utilizing Indonesian population data for initial values. The basic reproduction number was estimated at $\mathcal{R}_0 \approx 2.0$, consistent with early COVID-19 transmission estimates. The simulation generated a clear epidemic wave that peaked sharply around day 100 before rapid subsidence, validating the 80% mild case assumption. Long-term analysis showed that the sequential three-dose strategy causes the V_3 compartment to become the dominant immune group, while the susceptible population (S) stabilizes at a minimal

*Corresponding author

E-mail address: robynirawan.tjia@unpar.ac.id

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level. The model predicts a successful transition toward an endemic state. These findings highlight the critical role of disease modelling in assessing the effectiveness of interventions made to prevent further outbreaks of a disease.

Keywords: SEQHRV3 model; vaccination; epidemic; community immunity; COVID-19.

2020 AMS Subject Classification: 92D30, 34A34, 37N25.

1. INTRODUCTION

As the worldwide pandemic drew to a close, Coronavirus Disease (COVID-19) left a significant number of lessons to be learned. This airborne infection is caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which primarily leads to upper to lower respiratory tract dysfunction with symptoms ranging from mild to severe [1]. Individuals infected with this RNA virus are at an extremely high risk of person-to-person transmission. Close contact and social activities that generate aerosols created an ideal environment for the disease's spread, exponentially escalating the disease curve and triggering a global outbreak [2]. Data indicated that COVID-19 was a highly virulent infection capable of infecting up to 70% of the population without the development of herd immunity [3]. COVID-19 itself displayed a unique epidemiological pattern, characterized by multiple peaks in its epidemic curve between 2019 and 2022, largely attributed to viral mutations [4]. Interestingly, without effective intervention, a community risked losing vital daily social interactions as a consequence of these diseases. Several previous studies accommodating the factors potentially affected by the pandemic have been conducted, providing a compelling rationale that anticipating the spread of a disease with pandemic potential is an imperative to mitigate further casualties [5].

Similar viral outbreaks have, in fact, occurred at least twice over the past two decades, although generally on a more localized scale [6]. According to the World Health Organization's (WHO) most recent list of priority pathogens, it is highly probable that COVID-19 will not be the last pandemic, thereby necessitating heightened global vigilance [7]. Several other viruses within the Coronaviridae family (e.g., MERS-CoV, SARS-CoV) have already garnered serious attention due to their notable pathogenicity [8, 7]. Furthermore, other pathogens, such as the H5N1 influenza virus and Human Metapneumovirus (HMPV), which exhibit high mutation rates and airborne transmission potential, also pose significant future threats and could potentially trigger subsequent outbreaks [9, 7]. Ultimately, this underscores the critical importance

of developing proactive strategies to mitigate the risks associated with emerging infectious diseases in the future, particularly within the context of an outbreak.

Disease modeling is crucial for the formulation of public health policies, particularly when responding to the nascent phases of an epidemic [10]. Prior to the COVID-19 pandemic, mathematical models were widely employed to analyze the dynamics of infectious diseases such as Ebola, Influenza, and Dengue Fever [11, 12, 13, 14, 15, 16]. These models offer a vital reference point for evaluating intervention strategies and transmission prevention measures. For instance, an influenza model was constructed to assess the impact of adaptive immune response during a viral infection, which is a critical parameter in combating the disease [11]. During the 2014 Ebola outbreak, mathematical modeling proved useful in evaluating the quarantine strategy, which was ultimately found to be less effective in controlling disease spread when applied in isolation [12]. In the context of dengue fever modeling, the inclusion of spatio-temporal data and social aspects of the community became essential inputs for generating more relevant outbreak predictions, allowing for the development of more pertinent policy drafting [13, 14, 15, 16]. These examples collectively highlight the necessity of a flexible modeling framework that can be rapidly implemented during an outbreak, thus enabling balanced decision-making regarding the implementation of public health policies, for example vaccination [17] and movement restriction measures [18].

Epidemiological models constitute a key mathematical framework utilized to analyze the impact of disease spread. The fundamental epidemiological model commonly used to represent disease dissemination is the SIR model [19], where S , I , and R sequentially denote the Susceptible, Infected, and Recovered groups of individuals. A critical quantity derived from such models is the basic reproduction number ($\mathcal{R}_0$), which represents the average number of secondary cases produced by one infectious individual in a fully susceptible population [20]. When $\mathcal{R}_0 > 1$, the disease grows in the population; when $\mathcal{R}_0 < 1$, it declines and eventually fades out. Studies have also shown that differences in model structure and data quality can substantially affect the accuracy of epidemic projections [21]. When COVID-19 emerged, various compartmental models were rapidly adapted to characterize its dynamics. Giordano et al. [22] proposed the SIDARTHE model, which distinguishes between different infection stages

and detection statuses and evaluated population-wide interventions in Italy. Ndairou et al. [23] incorporated a superspreader compartment and demonstrated how this group disproportionately amplifies transmission. Kucharski et al. [24] used stochastic modeling to estimate early SARS-CoV-2 transmission dynamics, while Bertozzi et al. [25] reviewed the main challenges in COVID-19 forecasting, including the substantial role of asymptomatic carriers estimated at around 40–45% in some settings [26]. Other compartmental models applied to COVID-19 further confirmed that quarantine-focused interventions are effective at reducing epidemic peaks when deployed early [27, 28, 29].

Epidemiological models that explicitly consider vaccination are proposed by Rahman et al. [30] and several other researchers [31, 32]. The SLMCRV₁V₂V₃ model proposed by Rahman et al. [30], which classifies individuals as Susceptible, Latent, Mild, Critical, Recovered, Vaccinated 1, Vaccinated 2, and Vaccinated 3, evaluated the impact of three-dose COVID-19 vaccination in Australia. Moore et al. [32] similarly demonstrated that vaccination combined with non-pharmaceutical interventions substantially reduces hospitalizations compared to either strategy alone. Comparable vaccination-inclusive models have been applied to COVID-19 in Nigeria [33], Mexico [34], and South Africa [35], consistently confirming that vaccination is the most effective long-term epidemic control strategy. Beyond epidemic suppression, Olaniyi et al. [36] further demonstrated through optimal control analysis that well-designed vaccination programs can substantially minimize both infection burden and associated economic costs. Ghostine et al. [31] projected that high vaccine coverage dramatically accelerates the decline of the susceptible fraction. A key structural feature in these models is the explicit inclusion of breakthrough infection risk parameters, reflecting the empirical finding that vaccines reduce but do not eliminate the risk of infection [30, 32].

This study aims to assess the modified SIR model, the SEQHRV3 model, in the context of the COVID-19 pandemic to give insight into the disease outbreak pattern while considering vaccination to control the disease's dissemination. The SEQHRV3 model consists of compartments for **S**usceptible (S), **E**xposed (E), **Q**uantined (Q), **H**ospitalized (H), **R**ecovered (R), **V**accine 1 (V_1), **V**accine 2 (V_2), and **V**accine 3 (V_3). Compared to existing vaccination models [31, 32], the SEQHRV3 model incorporates dose-specific breakthrough infection risk parameters (ρ_1 ,

ρ_2, ρ_3) for each sequential dose, calibrated to empirically reported effectiveness values, and is applied directly to Indonesian national data from BPJS Kesehatan (Indonesia's national health insurance program), the Ministry of Health's vaccination database, and World Bank mortality statistics.

To support the simulation of disease spread modelling using the SEQHRV3 model, relevant data were identified to determine the parameter values for the model. Subsequently, the simulation results from the model were analysed using these parameter values. The basic reproduction number is also derived analytically to provide an epidemiological threshold for interpreting the simulation. It is anticipated that these simulation results will be useful for providing insight into the spread of a disease while considering vaccination, thereby facilitating the implementation of anticipatory measures to control the disease's dissemination.

2. MATERIALS AND METHODS

This research employs the SEQHRV3 disease spread model, modified from Rahman et al. [30], which accommodates vaccination compartments 1 through 3. The model was simulated using COVID-19 data. This disease was selected as a case study due to its status as a former pandemic from 2020 to 2021 and the availability of comprehensive data required to support the modeling in this research (Infeksi Emerging Kemenkes, accessed November 2025). Key parameters influencing the disease's spread were identified from the SEQHRV3 model. Furthermore, specific parameters influencing disease transmission under the influence of vaccinations 1 through 3 were also identified.

The schematic diagram of the SEQHRV3 disease spread model is presented in Figure 1. The SEQHRV3 model sequentially defines the compartments as Susceptible (S), Exposed (E), Mildly infected and then Quarantined (Q), Critically infected and then Hospitalized (H), and Recovered (R). Furthermore, the compartmental model focuses on the implementation of vaccination, leading to the addition of individual groups, namely those who have received the first vaccine dose (V_1), the second dose (V_2), and the third dose (V_3). In each transition process of individuals from one compartment to another, there are assumed parameters that influence the number of individuals migrating. The term ASK used in Table 1 refers to the national health

insurance scheme of Indonesia (Asuransi Sosial Kesehatan), administered by BPJS Kesehatan.

Descriptions of compartments and parameters are explained in Tables 1 and 2, respectively.

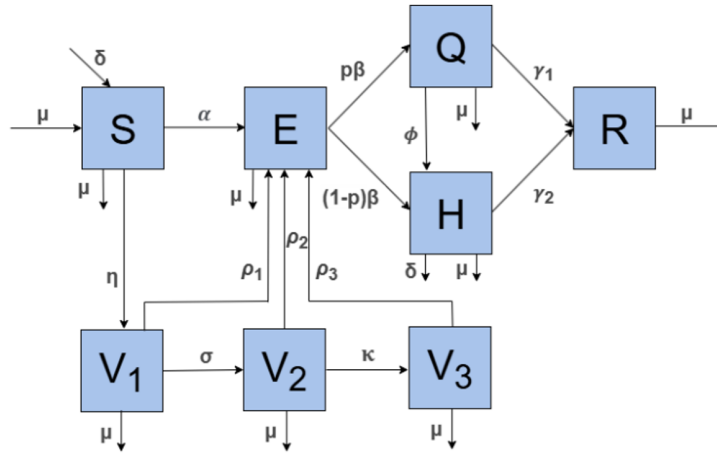


FIGURE 1. SEQHRV3 compartmental model diagram

Table 1: Compartment descriptions.

Compartment	Description
S (Susceptible)	The number of susceptible individuals enrolled in the ASK (BPJS Kesehatan) national health insurance program
E (Exposed)	The number of exposed individuals who are already infected and capable of transmitting the disease but remain asymptomatic (pre-symptomatic)
Q (Quarantined)	The number of individuals with mild symptoms undergoing self-isolation
H (Hospitalized)	The number of individuals with severe symptoms in hospital care

Continued on next page

Table 1 – continued from previous page

Compartment	Description
V_1 (Vaccinated 1)	The number of individuals vaccinated with the first dose
V_2 (Vaccinated 2)	The number of individuals vaccinated with the second dose
V_3 (Vaccinated 3)	The number of individuals vaccinated with the third dose
R (Recovered)	The number of individuals who have recovered from the disease

Table 2: Parameter descriptions.

Parameter	Description	Unit
μ	The ASK participation rate, assumed equal to the natural mortality rate	day^{-1}
δ	The mortality rate from COVID-19 among hospitalized patients	day^{-1}
α	The rate of disease transmission	day^{-1}
β	The virus incubation rate (inverse of incubation period)	day^{-1}
η	First-dose vaccination coverage rate	day^{-1}
σ	Second-dose vaccination coverage rate	day^{-1}
κ	Third-dose vaccination coverage rate	day^{-1}
ρ_1	Breakthrough infection risk rate after the first vaccine dose	day^{-1}

Continued on next page

Table 2 – continued from previous page

Parameter	Description	Unit
ρ_2	Breakthrough infection risk rate after the second vaccine dose	day ⁻¹
ρ_3	Breakthrough infection risk rate after the third vaccine dose	day ⁻¹
p	Proportion of cases with mild or asymptomatic symptoms	dimensionless
ϕ	Hospitalization rate among self-quarantined individuals	day ⁻¹
γ_1	Recovery rate among self-quarantined individuals	day ⁻¹
γ_2	Recovery rate among hospitalized individuals	day ⁻¹

If N represents the total population and $t \geq 0$ denotes the time in days, then N can be expressed as

$$(1) \quad N = S(t) + E(t) + Q(t) + H(t) + R(t) + V_1(t) + V_2(t) + V_3(t).$$

Based on the compartmental model illustrated in Figure 1, the following system of differential equations is formulated using initial values $S(0) \geq 0$, $E(0) \geq 0$, $Q(0) \geq 0$, $H(0) \geq 0$, $R(0) \geq 0$, $V_1(0) \geq 0$, $V_2(0) \geq 0$, and $V_3(0) \geq 0$:

$$\begin{aligned} \frac{dS(t)}{dt} &= \mu N + \delta H(t) - \frac{\alpha S(t)E(t)}{N} - S(t)(\eta + \mu), \\ \frac{dE(t)}{dt} &= \frac{\alpha S(t)E(t)}{N} + \frac{\rho_1 V_1(t)E(t)}{N} + \frac{\rho_2 V_2(t)E(t)}{N} \\ &\quad + \frac{\rho_3 V_3(t)E(t)}{N} - E(t)(\beta + \mu), \\ \frac{dQ(t)}{dt} &= p\beta E(t) - Q(t)(\gamma_1 + \mu + \phi), \end{aligned}$$

$$\begin{aligned}
(2) \quad \frac{dH(t)}{dt} &= (1-p)\beta E(t) + \phi Q(t) - H(t)(\gamma_2 + \mu + \delta), \\
\frac{dR(t)}{dt} &= \gamma_1 Q(t) + \gamma_2 H(t) - \mu R(t), \\
\frac{dV_1(t)}{dt} &= \eta S(t) - \frac{\rho_1 V_1(t)E(t)}{N} - V_1(t)(\sigma + \mu), \\
\frac{dV_2(t)}{dt} &= \sigma V_1(t) - \frac{\rho_2 V_2(t)E(t)}{N} - V_2(t)(\kappa + \mu), \\
\frac{dV_3(t)}{dt} &= \kappa V_2(t) - \frac{\rho_3 V_3(t)E(t)}{N} - \mu V_3(t).
\end{aligned}$$

In Eq. (2), μN represents the number of individuals entering the S compartment. The notation $\delta H(t)$ represents the number of individuals dying from COVID-19 among hospitalized individuals. To maintain the population size N constant within this compartmental model, $\delta H(t)$ is assumed to re-enter the S compartment. The notation $\alpha S(t)E(t)/N$ represents the incidence number of susceptible individuals who interact with exposed individuals. The terms $\rho_1 V_1(t)E(t)/N$, $\rho_2 V_2(t)E(t)/N$, and $\rho_3 V_3(t)E(t)/N$ represent breakthrough infections among first-, second-, and third-dose recipients, respectively.

The notation $\eta S(t)$ denotes the number of susceptible individuals receiving the first vaccination dose. The notations $\sigma V_1(t)$ and $\kappa V_2(t)$ denote the number of individuals who received the second and third vaccination doses, respectively. The notation $p\beta E(t)$ represents the number of exposed individuals with mild symptoms undergoing self-quarantine, and $(1-p)\beta E(t)$ represents those with severe symptoms requiring hospitalization. The notation $\gamma_1 Q(t)$ represents the number of individuals recovering from self-quarantine, and $\gamma_2 H(t)$ represents the number recovering from hospitalization. Finally, $\phi Q(t)$ represents the number of self-quarantined individuals whose condition worsens, thus requiring hospitalization.

Rapid prevention of disease spread during a pandemic can be achieved, notably by deploying vaccines to susceptible populations. Data on COVID-19 in Indonesia during this pandemic period indicate that the dissemination occurred very rapidly in mid-2021 and early 2022, evident from the spike in the graph of confirmed COVID-19 cases in Figure 2. It is expected that if a vaccine can be discovered more quickly, the spread of a disease during a pandemic can be

suppressed more rapidly. Therefore, there is a need for compartments that accommodate the vaccine factor in the process of handling the ongoing outbreak.

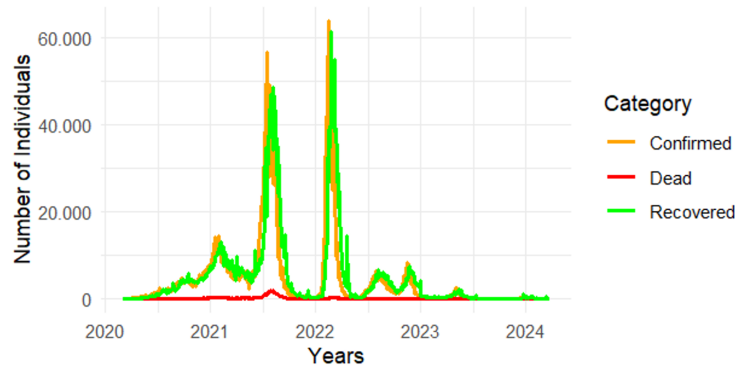


FIGURE 2. COVID-19 trend data for Indonesia (2020–2024). Source: Kemenkes.

The following details the data utilized for the simulation. First, to estimate the natural mortality rate parameter (μ), annual mortality rate data per 1,000 individuals in Indonesia from 2015 to 2019 (World Bank, accessed March 2025) were used, as summarized in Table 3. The next data set comprises the number of individuals receiving each vaccine dose, used to estimate the vaccination coverage rate parameters η , σ , and κ (Kemenkes, accessed November 2025), as shown in Table 4. The initial number of susceptible individuals (S_0) was assumed to be the total number of participants in Indonesia’s BPJS Kesehatan in 2024, equal to 277,538,004 individuals.

TABLE 3. Mortality rate data for Indonesia (2015–2019). Source: World Bank, accessed March 2025.

Year	Mortality rate (per 1,000 individuals)
2015	7.460
2016	7.529
2017	7.579
2018	7.496
2019	7.539

TABLE 4. Number of individuals who received each COVID-19 vaccine dose.

Source: Kemenkes, accessed November 2025.

Vaccine dose	Number of individuals
First dose	241,125,014
Second dose	206,960,090
Third dose	108,572,867

3. RESULTS AND DISCUSSION

This section details the method for determining the values of the parameters required for the model simulation. The explanation is followed by the presentation of the solution to the system of differential equations derived from Eq. (2). Subsequently, the graphs of the resulting solution are displayed and the findings are analysed.

3.1. Parameter Values Determination. The value of μ , the natural mortality rate, is assumed to be equal to the average of the mortality rate data presented in Table 3 and subsequently converted into a daily rate. Assuming the average annual mortality rate per 1,000 individuals is 7.5206, the resulting value for μ per day is:

$$\mu = \frac{7.5206}{1,000 \times 360} = 2.0891 \times 10^{-5} \text{ day}^{-1}.$$

To determine the daily disease transmission rate, denoted as α , reference is made to the COVID-19 transmission rate reported by Zhao et al. [37], which is $\alpha = 0.25 \text{ day}^{-1}$. The daily viral incubation rate $\beta = 0.125 \text{ day}^{-1}$ is assumed based on the virus incubation period cited from the Ciputra Hospital website. To determine the values of η , σ , and κ , the following equation is utilized:

$$g_i = \frac{V_i}{V_{i-1} \cdot T},$$

where V_i is the number of individuals receiving the i -th vaccine dose ($i \in \{1, 2, 3\}$), V_0 is assumed to be equal to S_0 , and T denotes the vaccination schedule, assumed to be one year (360 days). Based on Table 4, the calculated values are:

$$\eta = \frac{241,125,014}{277,538,004 \times 360} = 2.413 \times 10^{-3} \text{ day}^{-1},$$

$$\sigma = \frac{206,960,090}{241,125,014 \times 360} = 2.384 \times 10^{-3} \text{ day}^{-1},$$

$$\kappa = \frac{108,572,867}{206,960,090 \times 360} = 1.457 \times 10^{-3} \text{ day}^{-1}.$$

The determination of the parameter values ρ_1 , ρ_2 , and ρ_3 uses the formula:

$$\rho_y = \frac{1 - \lambda_y}{\tau},$$

where λ_y is the effectiveness of the y -th vaccine dose and τ is the assumed duration of vaccine effectiveness in days. The values for λ_1 and λ_2 were sourced from PubMed Central (NCBI), and λ_3 was obtained from EurekaAlert! (AAAS). These represent the effectiveness of the first, second, and third vaccine doses at 76%, 82%, and 93%, respectively. The duration of vaccine effectiveness is assumed to be 360 days ($\tau = 360$). Therefore:

$$\rho_1 = \frac{1 - 0.76}{360} \approx 6.7 \times 10^{-4} \text{ day}^{-1},$$

$$\rho_2 = \frac{1 - 0.82}{360} \approx 5.0 \times 10^{-4} \text{ day}^{-1},$$

$$\rho_3 = \frac{1 - 0.93}{360} \approx 1.9 \times 10^{-4} \text{ day}^{-1}.$$

The parameter p , representing the proportion of mild or asymptomatic cases, is assumed to be 80% based on WHO data. Several parameters, namely δ , ϕ , γ_1 , and γ_2 , lack supporting source data and were therefore self-assumed to produce a simulation closely approximating real conditions: $\delta = 0.01 \text{ day}^{-1}$, $\phi = 0.05 \text{ day}^{-1}$, $\gamma_1 = 0.1 \text{ day}^{-1}$, and $\gamma_2 = 0.08 \text{ day}^{-1}$. All parameter values are summarized in Table 5.

3.2. Basic Reproduction Number. The basic reproduction number ($\mathcal{R}_0$) is a key epidemiological threshold that determines whether an epidemic will grow or decline in a fully susceptible population [20]. To derive $\mathcal{R}_0$ for the SEQHRV3 model, the next-generation matrix (NGM) method is applied at the disease-free equilibrium (DFE), where $S = N$ and all other compartments equal zero. At the DFE, new infections arise only from the E compartment through the term $\alpha S(t)E(t)/N$, with outflow rate $(\beta + \mu)$. Applying the NGM method [20]:

$$(3) \quad \mathcal{R}_0 = \frac{\alpha}{\beta + \mu}.$$

TABLE 5. Parameter values used in the SEQHRV3 model simulation.

Parameter	Value	Unit	Source
μ	2.0891×10^{-5}	day ⁻¹	Table 3
δ	0.01	day ⁻¹	Assumed
α	0.25	day ⁻¹	Zhao et al. [37]
β	0.125	day ⁻¹	Ciputra Hospital
η	2.413×10^{-3}	day ⁻¹	Table 4
σ	2.384×10^{-3}	day ⁻¹	Table 4
κ	1.457×10^{-3}	day ⁻¹	Table 4
ρ_1	6.7×10^{-4}	day ⁻¹	NCBI and AAAS
ρ_2	5.0×10^{-4}	day ⁻¹	NCBI and AAAS
ρ_3	1.9×10^{-4}	day ⁻¹	NCBI and AAAS
p	0.8	dimensionless	WHO
φ	0.05	day ⁻¹	Assumed
γ_1	0.1	day ⁻¹	Assumed
γ_2	0.08	day ⁻¹	Assumed

Substituting the parameter values gives $\mathcal{R}_0 = 0.25 / (0.125 + 2.0891 \times 10^{-5}) \approx 2.0$. This is consistent with early COVID-19 $\mathcal{R}_0$ estimates in the literature, which ranged from approximately 1.4 to 3.9 [3, 24, 37]. The corresponding herd immunity threshold is $\text{HIT} = 1 - 1/\mathcal{R}_0 = 0.5$, meaning at least 50% of the population must acquire immunity to prevent sustained transmission.

3.3. Solution of the Differential Equation System for the SEQHRV3 Model. To facilitate understanding of the procedure employed in this study, the steps involved in simulating the SEQHRV3 model are illustrated in the flow chart shown in Figure 3.

In order to find the solution to the system of differential equations and to plot the simulation results, initial values for all compartments are required. The initial value for the S compartment

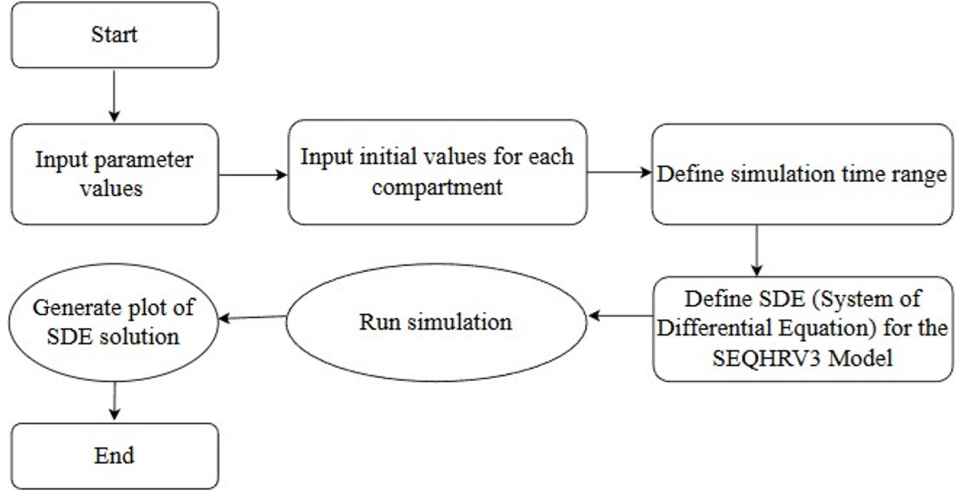


FIGURE 3. SEQHRV3 model simulation flow chart.

is:

$$s(0) = \frac{S(0)}{N} = \frac{277,538,004}{281,604,000} \approx 0.986,$$

where $N = 281,604,000$ is the total population of Indonesia in 2024, obtained from Statistics Indonesia (BPS). The initial value for the E compartment is determined by assuming $E(0) = 10,000$, yielding:

$$e(0) = \frac{E(0)}{N} = \frac{10,000}{281,604,000} \approx 3.551 \times 10^{-5}.$$

The proportion of cases with severe symptoms requiring hospitalization is assumed to be 20%, giving $H(0) = 2,000$ and:

$$h(0) = \frac{H(0)}{N} = \frac{2,000}{281,604,000} \approx 7.102 \times 10^{-6}.$$

The initial value for the Q compartment, based on the 80% mild case assumption, is:

$$q(0) = \frac{Q(0)}{N} = \frac{8,000}{281,604,000} \approx 2.841 \times 10^{-5}.$$

No individuals are assumed to have recovered or been vaccinated at $t = 0$, so $r(0) = v_1(0) = v_2(0) = v_3(0) = 0$. The solution to the system in Eq. (2) is presented graphically, displaying compartments e , q , h , and r in Figure 4, and compartments s , v_1 , v_2 , and v_3 in Figure 5. This separation was performed to ensure clarity in the visualization, as the scale of the e , q , h , and r compartments is significantly smaller compared to Figure 5.

3.4. Analysis of the Epidemic Wave. The analysis conducted in this section is based on the solution results of the e , q , h , and r compartments over a period of 360 days, as shown in Figure 4.

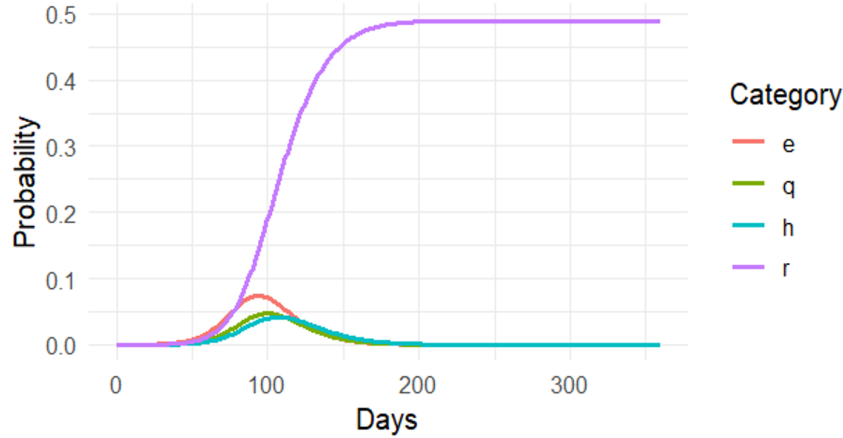


FIGURE 4. Solution of the SEQHRV3 model ODE system for the e , q , h , and r compartments over 360 days.

The simulation demonstrates a clear epidemic wave pattern. The probability of individuals being in compartments e , q , and h (which represent active cases) starts near zero, undergoes a sharp increase, and reaches its peak at approximately day 100. The peak of the e curve (exposed) is the highest among infectious cases, followed by q (self-quarantine), and finally h (hospitalized).

Regarding the Quarantine vs. Hospitalized ratio, the significantly higher peak of q compared to h is consistent with the model's assumption where the proportion of mild or asymptomatic cases (p) was set at 80% ($p = 0.8$). This reflects the real-world situation where the majority of COVID-19 cases undergo self-quarantine, and only a small fraction (those with severe symptoms) require hospitalization [26]. This pattern is also consistent with results reported in COVID-19 modeling studies conducted for other country settings, where a high proportion of mild cases substantially reduces hospitalization burden while widening the epidemic base [27, 28, 29].

After reaching their peaks, the three case curves (e , q , h) experience a sharp decline and return close to zero after approximately day 200. Concurrently with the subsidence of the case wave, the r curve (recovered) shows a significant increase, beginning around day 50 and becoming

constant after day 150. This indicates the accumulation of individuals who have recovered from the infection and are assumed to have immunity.

It is also important to critically interpret the discrepancy between the single epidemic wave generated by the simulation (Figure 4) and the multi-wave pattern observed in the actual Indonesian COVID-19 data (Figure 2). The simulation results, derived from a set of fixed parameters, depict an idealized outbreak trajectory that rises and falls based on initial transmission conditions and vaccination rates alone. In contrast, the real-world data exhibits multiple distinct peaks, particularly the significant surges recorded in mid-2021 and early 2022. This deviation is primarily attributable to the dynamic nature of the pandemic, which involves variables not captured in a static parameter model. Specifically, the emergence of novel variants of concern (such as Delta and Omicron) drastically altered the transmission rate (α) and the vaccine escape potential (ρ) over time [4]. COVID-19 modeling studies have similarly noted this limitation of fixed-parameter models and have proposed time-varying parameter extensions to better capture variant-driven surges [22, 24].

Additionally, the implementation of non-pharmaceutical interventions (NPIs), such as the Community Activities Restrictions Enforcement (PPKM) in Indonesia, introduced time-varying fluctuations in social contact rates [18]. Therefore, while this model successfully quantifies the fundamental impact of sequential vaccination on suppressing a primary wave, future iterations incorporating time-dependent parameters would be required to reconstruct the complex, multi-wave historical trends driven by viral evolution and changing government policies [21, 25].

3.5. Analysis of Population and Vaccination Dynamics. The analysis conducted in this section is based on the solution results of the s , v_1 , v_2 , and v_3 compartments with a simulation duration of 5,000 days, as shown in Figure 5.

It can be seen that there is a decline in the susceptible population. The s curve shows a drastic decrease in probability from its initial value (≈ 0.986), particularly during the first 150 days. This decline is attributed to two main factors: individuals becoming infected (moving to the e compartment) and individuals receiving the first vaccine dose (moving to the v_1 compartment). Moreover, Figure 5 provides a more comprehensive overview. The v_1 curve (Dose 1) reaches its peak around day 500, then declines as individuals transition to receive the second dose. The

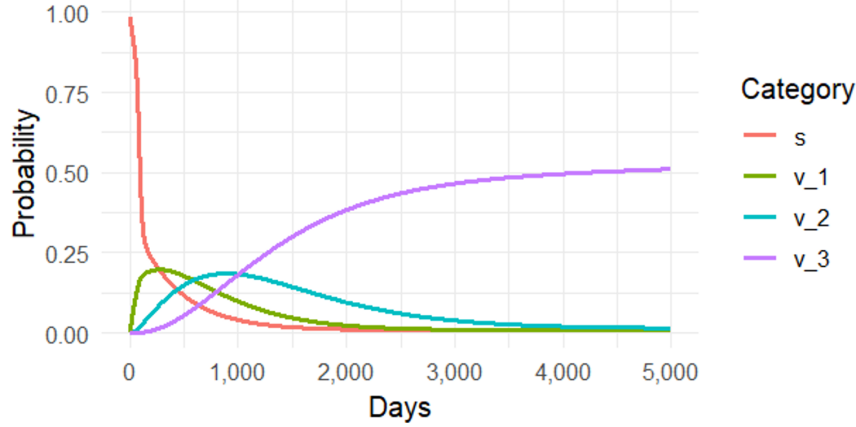


FIGURE 5. Solution of the SEQHRV3 model ODE system for the s , v_1 , v_2 , and v_3 compartments over 5,000 days.

v_2 curve (Dose 2) exhibits a similar pattern, peaking around day 1,000, followed by a decline as individuals transition to receive the third dose. The v_3 curve (Dose 3) demonstrates a slow but steady increase, eventually becoming the dominant vaccination compartment in the population. These sequential peak patterns are directly produced by the model's structure, consistent with findings from related vaccination-inclusive compartmental models applied to other national settings [30, 31].

In the long term, the simulation shows that the susceptible population (s) stabilizes at a very low level, while the majority of the population resides in the v_3 (fully vaccinated) and r (recovered) compartments. This illustrates the population's shift toward community immunity, achieved through both natural infection and vaccination as a result of disease spread and intervention. The declining breakthrough infection rates across doses ($\rho_1 > \rho_2 > \rho_3$) ensure that individuals in V_3 carry the lowest residual infection risk, which is consistent with empirical evidence that booster doses substantially reduce breakthrough infection probability [32]. These long-term dynamics are comparable to results reported by Ghostine et al. [31] and Moore et al. [32], where high vaccine coverage with multiple doses was shown to be the most effective long-term strategy for minimizing the susceptible fraction.

3.6. Model Limitations and Recommendations. Several limitations of the SEQHRV3 model should be noted. First, four parameters (δ , ϕ , γ_1 , γ_2) were self-assumed due to the

absence of supporting source data for these specific rates in the Indonesian COVID-19 context. Sensitivity analyses on these parameters are recommended to assess their influence on the simulation outcomes. Second, the model does not account for waning immunity in vaccinated or recovered individuals; in practice, protection from both natural infection and vaccination declines over time, particularly against new variants [4]. Third, the population is treated as homogeneous, ignoring age structure, geographic distribution, and socioeconomic disparities in vaccine access. Fourth, NPIs such as mobility restrictions, which significantly shaped the Indonesian epidemic curve, are not represented in the model [18].

To enhance the robustness of the SEQHRV3 model, integrating time-dependent parameters for α and ρ is recommended to capture the effects of variant emergence and changing public behavior [22, 24]. Adding a waning immunity term to the vaccination and recovery compartments would better reflect real-world immunological dynamics [32]. Furthermore, exploring different vaccination scheduling scenarios is crucial to identifying the most efficient strategy for epidemic suppression, and extending the model to incorporate economic variables would support comprehensive cost-benefit evaluations of public health interventions.

4. CONCLUSIONS

The simulation results of the SEQHRV3 model confirm the expected dynamics of a pandemic controlled by a sequential vaccination program. The basic reproduction number $\mathcal{R}_0 \approx 2.0$, derived analytically using the next-generation matrix method, is consistent with early COVID-19 literature estimates [3, 24, 37] and confirms that the epidemic grows in a fully susceptible population but is controllable through combined vaccination and isolation. The long-term analysis demonstrated the effectiveness of the three-dose policy. While initial vaccination compartments (V_1 , V_2) were transient, the V_3 compartment eventually became the dominant immune group, leading to a long-term stabilization. Over 5,000 days, the population stabilized, with the susceptible fraction (s) becoming minimal. This indicates a successful transition toward an endemic state driven by high levels of community immunity (from both V_3 and R compartments). The study underscores the necessity of rapid and multi-dose vaccination deployment as a highly effective anticipatory measure against disease spread. Furthermore, the model can be utilised to project epidemic waves characterized by similar disease dissemination patterns.

To enhance the robustness of the SEQHRV3 model, integrating time-dependent parameters is recommended to model the decay of long-term immunity, thereby reflecting real-world immunological dynamics. Furthermore, exploring different vaccination scheduling scenarios is crucial to identifying the most efficient strategy for rapid epidemic suppression and maximizing community immunity. Another recommendation is to evaluate vaccine effectiveness for other diseases exhibiting similar dissemination patterns and to consider the integration of additional interventions if necessary. The modelling approach can also be extended to incorporate economic variables for a comprehensive cost-benefit evaluation of public health interventions.

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CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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