

THE IMPACT OF BOOSTER VACCINE ON THE SPREAD OF COVID-19 USING AN EXTENDED SEIR MODEL

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Abstract

This study used a non-linear SEIR model to analyze the spread of COVID-19 in Malaysia. The model considered vaccines, booster doses, hospitalized cases, and various infectious cases. The model was fitted to real data using MATLAB's ode solver and the particle swarm optimization method. The results revealed that booster vaccines significantly reduced the total number of infected cases by 62% when 70% of the population received them. This model can help policymakers to make crucial decisions to prevent future outbreaks.

Key words: booster vaccine, mathematical model, reproduction number, optimization, sensitivity analysis

2020 Mathematics Subject Classification: 34C60, 92C60

Introduction. In late December 2019, the coronavirus disease (COVID-19) emerged in Wuhan, China, and has posed significant global health and economic threats [1,2]. As of November 20, 2023, there have been over 5.13 million cases with 37 000 deaths in Malaysia. ARORA et al. [3] found that many infected individuals had underlying health conditions like hypertension and diabetes. To control the spread of COVID-19, Malaysia and other countries have implemented various measures such as movement control orders, travel restrictions, lockdowns, mandatory masks, social distancing, and isolation since early 2020 [2,4]. To control the

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COVID-19 virus in Malaysia, vaccination programmes were implemented in the first quarter of 2021 to gain herd immunity by reducing transmission and mortality rates. As of October 2021 in Klang Valley (KV), the majority received at least one dose (87.3%) or complete dose (81.4%) of the vaccine [5]. The booster vaccine programme began on October 13th for those who had already received complete doses. Along with medical and biological research, mathematical epidemiology models, including the compartmental Susceptible-Exposed-Infectious-Recovered (SEIR) model, are crucial for understanding and forecasting infectious diseases like measles, dengue, and COVID-19 [6–9]. Various methods and machine learning techniques have also been utilized to analyze disease transmission dynamics and evaluate interventions such as social distancing, quarantine, vaccination, contact tracing, and travel restrictions on COVID-19 [2, 7, 8].

This study proposes a unique SEIR model that includes compartments such as vaccination and booster campaigns, hospitalized critical cases, and refined exposed and infected population groups. Other studies featured models with some or all of these features; however, none included the early booster vaccine programme timeline in Malaysia or all current arrangements as in this paper. For example, GHOSTINE et al. [9] extended the SEIR model by the addition of vaccination, quarantine (isolation), and death, while investigating vaccine impact and sensitivity. The study of AZIZ et al. [7] incorporated several extensions to the SEIR model, such as interstate travel restrictions, two doses of vaccination, and refined exposed state in earlier stages of vaccination and post-travel restrictions. Similarly, RAJA et al. [8] used an extensive SEIR model with vaccination, symptomatic, asymptomatic, traced, and non-traced population groups, along with a sensitivity analysis of different parameters. MOORE et al. [10] highlighted the significance of multiple vaccine doses to reduce COVID-19 cases and fatalities. Results stated, that receiving three vaccine doses lowered the incidence of infection compared to those with one or two doses, supporting the widely accepted notion that a two or three-dose vaccine regimen enhances protection against COVID-19 for about 3 to 5 months [11]. This study developed a comprehensive ten-compartment SEIR model that captures the dynamics of COVID-19 and provides future projections, focusing on the impact of booster vaccinations in reducing virus transmission in Malaysia from October 1, 2021, to January 31, 2022.

Methodology. Data source. The study conducted a thorough analysis of the available data, focusing specifically on the COVID-19 situation in KV, Malaysia. The data cover the period from 1st October 2021 to 31st January 2022, capturing the start of the booster vaccine campaign [5, 12]. The data were extracted directly from the open-source website of the Ministry of Health of Malaysia¹ (MOH) containing information on daily active cases, vaccination rates, hospitalizations, and COVID-19-related deaths [13].

¹<https://github.com/MoH-Malaysia/covid19-public>

Extended SEIR model. In this research, we used the SEIR model and extended it to ten compartments followed by simulating and analyzing the trend of COVID-19 in Malaysia. The population was divided into ten major compartments: the population susceptible (S), fully vaccinated (V), booster vaccinated (B), exposed from susceptible (E_s), exposed from vaccinated (E_v), infectious (severe) (I_s), infectious (non-severe) (I_{ns}), recovered (R), hospitalized critical cases (H), and dead (D). In this model, we have the following assumptions: the total population in KV was assumed to be susceptible with at least one exposed or infected case for the process to begin, all model compartments were considered to be homogeneous without any changes due to demographic factors, individuals with incomplete vaccines were considered to be susceptible, referring to several articles like [3, 8–10], we assumed approximately 80% and 20% of E_s compartment go to I_s and I_{ns} compartments, respectively, and all individuals in R compartment were assumed to have achieved immunity from COVID-19.

The non-linear differential equations of extended SEIR model are as follows:

$$\begin{aligned} \frac{dS}{dt} &= b - \frac{\beta(t)S}{N} (I_s + I_{ns}) - \alpha(t)S - \rho S, \quad \frac{dV}{dt} = \alpha(t)S - \frac{\beta(t)\sigma_1 V}{N} (I_s + I_{ns}) - \mu(t)V - \rho V, \\ \frac{dB}{dt} &= \mu(t)V - \frac{\beta(t)\sigma_2 B}{N} (I_s + I_{ns}) - \rho B, \quad \frac{dE_s}{dt} = \frac{\beta(t)S}{N} (I_s + I_{ns}) - \gamma E_s - \rho E_s, \\ \frac{dE_v}{dt} &= \frac{\beta(t)\sigma_1 V}{N} (I_s + I_{ns}) + \frac{\beta(t)\sigma_2 B}{N} (I_s + I_{ns}) - \gamma\sigma_1 E_v - \gamma\sigma_2 E_v - \rho E_v, \quad \frac{dH}{dt} = \\ &\theta_4 I_s - \theta_3 H - \epsilon_2 H, \quad \frac{dI_s}{dt} = 0.8\gamma E_s + \gamma\sigma_1 E_v - \theta_1 I_s - \theta_4 I_s - \epsilon_1 I_s - \rho I_s, \quad \frac{dI_{ns}}{dt} = \\ &0.2\gamma E_s + \gamma\sigma_2 E_v - \theta_2 I_{ns} - \rho I_{ns}, \quad \frac{dR}{dt} = \theta_1 I_s + \theta_2 I_{ns} + \theta_3 H - \rho R, \quad \frac{dD}{dt} = \epsilon_1 I_s + \epsilon_2 H. \end{aligned}$$

The equations govern the model transmission, where N represents the total population in KV and the transmission rate is the function $\beta(t)$. Next, when in contact with E_s or E_v , any S , V , and B will experience a rate of infection of $\frac{\beta(t)S}{N}(I_s + I_{ns})$, $\frac{\beta(t)\sigma_1 V}{N}(I_s + I_{ns})$, and $\frac{\beta(t)\sigma_2 B}{N}(I_s + I_{ns})$, respectively. A death outflow, ρ exists for most compartments except compartments that account for deaths due to COVID-19. The γ denotes the incubation period of the system, with the daily average recovery and death rate denoted by θ_n ($n = 1, 2, 3, 4$), and ϵ_m ($m = 1, 2$), respectively. All the numerical simulations used MATLAB R2022b. The initial model parameter description, corresponding values, and sources used in the model are as follows: N is 8 417 800, daily natural birth rate, b is 1500, and daily natural death rate, ρ is 0.0000124. Data were obtained from the Department of Statistics Malaysia. The daily average values for booster vaccine efficacy, μ is 0.004432, transmission rate, β is 0.6529, disease-induced death rate, λ is 0.00038, and vaccination efficacy, α is 0.00096. Data were obtained from MOH [13]. Additionally, vaccine inefficacy, σ_1 is 0.20, booster inefficacy, σ_2 is 0.05, daily average rate of death from infected (severe) individuals, ϵ_1 is $\lambda/7$, and daily average rate of death from hospitalization, ϵ_2 is $\lambda/14$. Data were extracted from [7, 9]. Finally the daily average, recovery rate from infected (severe) individuals, θ_1 is $1/12.5$,

recovery rate from infected (non-severe) individuals, θ_2 is 1/10, recovery rate from hospitalization, θ_3 is 1/14, hospitalization rate from infected (severe) individuals, θ_4 is 1/5, and incubation rate, γ is 1/5. Data were extracted from [8].

Parameter optimization. The model simulation used real data to determine the optimized transmission and death rates due to COVID-19. Due to the complexity and variability of COVID-19, these rates fluctuate over time in discrete periods. The functions of β and λ were taken directly from [7] and both were calculated using the particle swarm optimization (PSO) algorithm in 10-day intervals. The MATLAB algorithms were retrieved and modified from AZIZ [14] and utilized the global optimal solution from PSO to fit the model.

Non-negativity of the model.

Theorem 1. *If all the initial conditions of the outlined parameters are greater than or equal to 0, then the solutions of the model equation remain non-negative for all $t > 0$ and within Ω , where $\Omega = ((S(t), V(t), B(t), E_s(t), E_v(t), I_s(t), I_{ns}(t), R(t), H(t), D(t)) \in \mathbb{R}_+^{10}, t > 0)$ represents the positive invariant, and all the solutions either approach or stay in Ω . Next, $\mathbb{R}_+^{10}$ is the non-negative octant and is positively invariant.*

Proof. To prove the system's non-negativity for all $t > 0$, every compartment's rate of change must be greater than the death outflow. From the model equations, we have $\frac{dS}{dt} \geq -(\alpha + \rho)S$ and by integrating it we receive $S(t) \geq S_0 e^{-(\alpha+\rho)t} \geq 0$. Hence, $S(t)$ remains non-negative for all $t > 0$. Similarly, the same derivation can be applied to other compartments showing variables to also be positive. For $I_s(t)$ we have, $\frac{dI_s}{dt} \geq -(\theta_1 + \theta_4 + \epsilon_1 + \rho)I_s$ and integrating it we get $I_s(t) \geq (I_s)_0 e^{-(\theta_1+\theta_4+\epsilon_1+\rho)t} \geq 0$. Likewise, all the variables are non-negative for all $t \geq 0$ proving the theorem. $\square$

Boundedness of the model.

Theorem 2. *If all variables of the proposed model have positive initial conditions, then the system is bounded within $0 \leq N(t) \leq \frac{b}{\rho N}$ for all $t \geq 0$.*

Proof. Summing up all the ODE equations of the model, we have the growth rate of the total population to be $\frac{dN}{dt} = \frac{dS}{dt} + \frac{dV}{dt} + \frac{dB}{dt} + \frac{dE_s}{dt} + \frac{dE_v}{dt} + \frac{dI_s}{dt} + \frac{dI_{ns}}{dt} + \frac{dR}{dt} + \frac{dH}{dt} + \frac{dD}{dt}$. By solving both sides of the equation we get $\frac{dN}{dt} = b - \rho N$ and when the limit of $t \rightarrow \infty$, we find $N(t) \leq \frac{b}{\rho N}$. As a result, from Theorem 2 and inequality of $N(t)$, we find that $0 \leq N(t) \leq \frac{b}{\rho N}$. Thus, all the variables $S(t)$, $V(t)$, $B(t)$, $E_s(t)$, $E_v(t)$, $I_s(t)$, $I_{ns}(t)$, $R(t)$, $H(t)$, and $D(t)$ are bounded. $\square$

Disease free equilibrium point. The disease-free equilibrium point (DFE) is represented by $\mathbf{X}^0$ of the transmission model acquired by setting the model

ODE equations, exposed and infected population to zero. We assume that $N = S^0 + V^0 + B^0$ hence, solving the ODE of the system we get the DFE,

$$\mathbf{X}^0 = \left(\frac{b}{(\alpha + \rho)}, \frac{\alpha b}{(\alpha + \rho)(\mu + \rho)}, \frac{\mu \alpha b}{\rho(\mu + \rho)(\alpha + \rho)}, 0, 0, 0, 0, 0, 0 \right),$$

where $\mathbf{X} = (S, V, B, E_s, E_v, I_s, I_{ns}, R, H, D)$. Next, the reproduction number, R_0 representing the average number of infections from infected individuals was derived. The R_0 depends on factors such as location, population density, and the next-generation matrix used to construct it [15]. The transmission and transition matrices were derived from the ODE of the infected states (E_s, E_v, I_s, I_{ns}) , resulting in a four-dimensional infected subsystem.

$$\mathbf{A} = \begin{pmatrix} 0 & 0 & \frac{\beta S^0}{N} & \frac{\beta S^0}{N} \\ 0 & 0 & \frac{\beta \phi}{N} & \frac{\beta \phi}{N} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \quad \text{and} \quad \Sigma = \begin{pmatrix} -a_1 & 0 & 0 & 0 \\ 0 & -a_2 & 0 & 0 \\ 0.8\gamma & \sigma_1\gamma & -a_3 & 0 \\ 0.2\gamma & \sigma_2\gamma & 0 & -a_4 \end{pmatrix},$$

where $a_1 = \gamma + \rho$, $a_2 = \sigma_1\gamma + \sigma_2\gamma + \rho$, $a_3 = \theta_1 + \theta_4 + \epsilon_1 + \rho$, $a_4 = \theta_2 + \rho$, $\phi = \sigma_1 V^0 + \sigma_2 B^0$. Next, the four-dimensional large domain of NGM is $K_L = -\mathbf{A}\Sigma^{-1}$. The basic NGM $\mathbf{K}$ is two-dimensional due to the two states of infection out of the four infected states as seen in $\mathbf{A}$. Using the epidemiological reasoning as stated by [16] to form the NGM, we construct the auxiliary matrix $\mathbf{E}$ with columns of two-unit vectors $(1, 0, 0, 0)'$ and $(0, 1, 0, 0)'$ and $\mathbf{K} = -\mathbf{E}'\mathbf{A}\Sigma^{-1}\mathbf{E}$ where $\det(\mathbf{K}) \neq 0$ allows no further reduction of dimensions. From the special case mentioned by DIEKMANN et al. [16], the transmission rate can also be written as $\beta = xy$, where x represents susceptibility and y represents the infectivity describing a contact tracing between two individuals. The separable mixing leads $\det(\mathbf{K}) = 0$ and therefore to $R_0 = \text{trace}(\mathbf{K})$ as $\mathbf{K}$ is two-dimensional. Proceeding with the special case to determine the NGM using the small domain K_S , we observe that the rows of the matrix $\mathbf{A}$ are multiple of vector $\mathbf{D} = (0, 0, y, y)$ and the columns are multiple of $\mathbf{C} = \left(\frac{xS^0}{N}, \frac{xS^0}{N}, 0, 0 \right)'$ with the note that $\mathbf{A} = \mathbf{C}\mathbf{D}$.

Then we write, $K_S = -\mathbf{D}\Sigma^{-1}\mathbf{C}$. Finally, the eigenvalue of the matrix K_S gives, $R_0 = \frac{\gamma\beta S^0}{Na_1} \left(\frac{0.8}{a_3} + \frac{0.2}{a_4} \right) + \frac{\beta\gamma\phi}{Na_2} \left(\frac{\sigma_1}{a_3} + \frac{\sigma_2}{a_4} \right)$.

Global stability of disease-free equilibrium point.

Theorem 3. *The DFE point $\mathbf{X}^0$ is globally asymptotically stable on Ω , for $V > \frac{\alpha S}{\mu + \rho}$ and $B > \frac{\mu V}{\rho}$.*

Proof. A Lyapunov function is constructed for the system, considering the function, as in [17]. $\mathbf{W}(t) = (S - S^0 \ln S) + (V - V^0 \ln V) + (B - B^0 \ln B) +$

$E_s + E_v + I_s + I_{ns} + R + H + D$, where S^0 , V^0 , and B^0 are the initial number of population in the respective group. Differentiating the function to time yields, $\dot{\mathbf{W}}(t) = -b(S - S^0)^2 - (V - V^0) \left(\frac{\alpha S}{V} - \frac{\alpha S^0}{V^0} \right) - (B - B^0) \left(\frac{\mu V^0}{B^0} - \frac{\mu V}{B} \right) - \rho(E_s + E_v + I_s + I_{ns} + R)$. By using LaSalle extension to Lyapunov's method [17], we assume $S > S^0$, $V > V^0$, $B > B^0$ and $0 < S < V < B < N$ then, $V > \frac{\alpha S}{\mu + \rho}$ and $B > \frac{\mu V}{\rho}$ hence, $\dot{\mathbf{W}}(t) \leq 0$ with consideration to $\mathbf{X}^0$. Finally, proving the DFE point $\mathbf{X}^0$ to be globally asymptotically stable on Ω . $\square$

Results and discussion. The extended SEIR model used to analyze and forecast the COVID-19 trend in KV, Malaysia, used the previously mentioned initial parameter values. The initial conditions of the model on 1st October 2021 ($t = 1$ day) include $V = 7004620$, $B = 118$, $I_s = 1859$, $I_{ns} = 24697$, $H = 178$, $D = 2671$, $S = N - V - B$ and the remaining compartments were set to zero.

Model fitting and projection. The model was fitted using the initial condition and real data. Figure 1 used 80% of the data for training and 20% for testing. The analysis focused on daily active cases, cumulative deaths, and cumulative booster vaccine administration. Figure 1a incorporates a time-varying transmission rate $\beta(t)$ and provides optimized model simulation using the particle swarm optimization method. This dynamic approach is necessary due to COVID-19's evolving behaviour over time. Figure 1(a-b) shows that model simulation closely matches the real data for active cases and cumulative deaths, with a spike in early 2022 during the projection due to factors such as the Omicron variant and inter-state travel component. In Fig. 1c, the simulation initially correlates strongly with actual booster dose administration, however, diverges gradually over time due to challenges in modelling recovery (immune population) and booster dose distribution (public from other states receiving booster in KV) which is similar to the vaccination compartment optimization simulation as shown by Aziz et al. [7]. The model's prediction capabilities were tested by optimizing techniques and fitting the model to real data, including daily infected cases, total fatalities, and booster doses in KV. Figure 1 is based on various variables such as COVID-19 testing capacity and the accuracy of reported data compared to the underestimated reported cases by other countries [7]. The PSO method helps achieve a closer fit between the simulated and real data as in Fig. 1(a-b).

Sensitivity analysis. To identify influential factors on different population groups within the model system and obtain a comprehensive understanding of the effects of infection, we varied several key parameters. Figure 2 represents the set of sensitivity analyses conducted on three population groups: Infected cases, Hospitalized, and Dead in columns 1, 2, and 3 respectively, while varying parameters like the initial number of booster vaccines, booster efficacy, and days to hospital admissions in rows 1, 2, and 3 respectively.

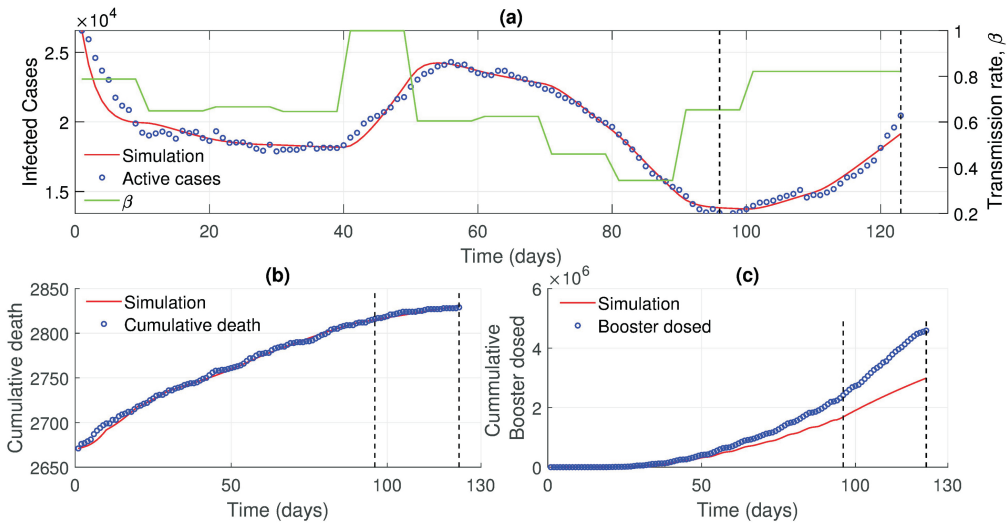


Fig. 1. Optimized model simulation with an 80% training data set and the projection with the remaining 20% testing data set

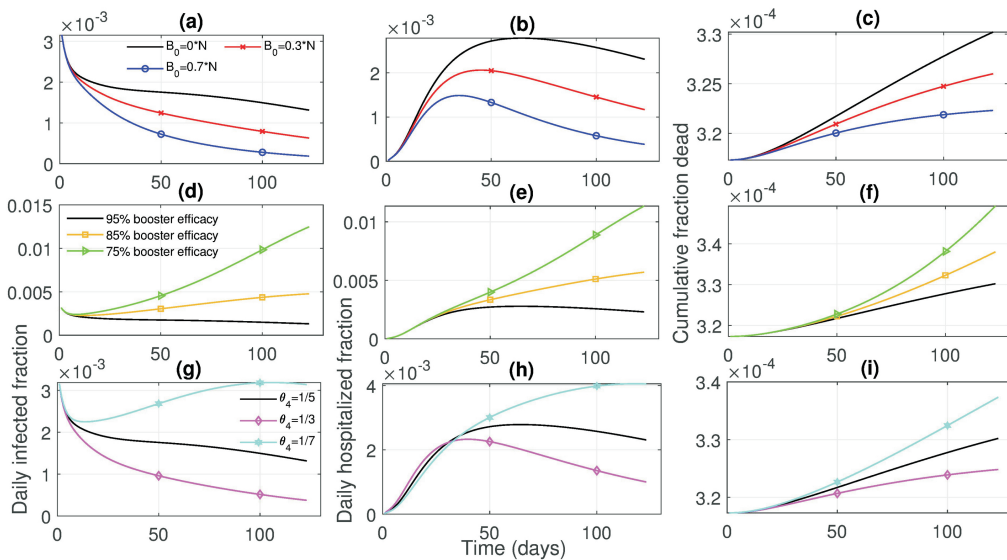


Fig. 2. Sensitivity analysis on three major population groups by varying key parameters

Results in Fig. 2(a–c) indicate a significant decrease in infected cases, hospitalizations, and death fraction with increased booster vaccination coverage. Compared to the population without booster vaccine, there was a reduction in total infected cases, daily hospitalization, and overall deaths by 62%, 47%, and 59%, respectively, with 70% booster vaccination coverage. These findings strongly support the positive impact of implementing booster vaccinations in achieving im-

munity and reducing disease-related fatalities. Figure 2(d–f) shows the impact of decreasing booster efficacy from 95% to 85% and 75%. With lower booster efficacy, there was an increase in infections, hospitalizations, and deaths. For instance, booster efficacy dropped from 95% to 75% leading to a four-fold increase in the total infected population, a five-fold increase in daily hospitalizations, and almost three times more COVID-19-related deaths. In brief, maintaining optimal booster effectiveness is crucial for anticipating and managing the availability of medical resources, as booster efficacy declines over time. Figure 2(h–i) demonstrates the impact of delayed admissions for severe cases, θ_4 . There was a noticeable correlation between the population fraction and changes in θ_4 . Early hospital admissions, $\theta_4 = 1/3$ for critical patients were found to be significant, while prolonged treatment for severely infected individuals led to a peak in infections and hospitalization on day 123 and a 54% increase in deaths. Therefore, prompt attention to severe infections by appropriate healthcare services is crucial to reduce transmission risks.

Conclusion. This study developed an expanded SEIR transmission model that integrates refined model compartments for a closer alignment with real data. The model was simulated under different scenarios through sensitivity analysis to investigate the impact of booster vaccination programmes. The results showed that booster vaccine programmes significantly control the spread of COVID-19, with consistent booster efficacy and quick hospital admission for severe infections. The model was also analytically verified to ensure desirable characteristics for simulating and predicting COVID-19 infection dynamics. It is worth noting that our results were based on the choice of parameter values. The sensitivity analysis was conducted in our study to ensure that the model performs adequately under a range of plausible parameter values. Furthermore, one can also fit the model to additional real datasets to improve the model’s predictions.

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