



Research paper

Mathematical model for understanding the relationship between diabetes and novel coronavirus

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ABSTRACT

A new model is proposed to explore interactions between diabetes and novel coronavirus. The model accounted for both the omicron variant and variants varying from omicron. The model investigated compartments such as hospitalization, diabetes, co-infection, omicron variant, and quarantine. Additionally, the impact of different vaccination doses is assessed. Sensitivity analysis is carried out to determine disease prevalence and control options, emphasizing the significance of knowing epidemics and their characteristics. The model is validated using actual data from Japan. The parameters are fitted with the help of "Least Square Curve Fitting" method to describe the dynamic behavior of the proposed model. Simulation results and theoretical findings demonstrate the dynamic behavior of novel coronavirus and diabetes mellitus (DM). Biological illustrations that illustrate impact of model parameters are evaluated. Furthermore, effect of vaccine efficacy and vaccination rates for the vaccine's first, second, and booster doses is conducted. The impact of various preventive measures, such as hospitalization rate, quarantine or self-isolation rate, vaccine dose-1, dose-2, and booster dose, is considered for diabetic individuals in contact with symptomatic or asymptomatic COVID-19 infectious people in the proposed model. The findings demonstrate the significance of vaccine doses on people with diabetes and individuals infectious with omicron variant. The proposed work helps with subsequent prevention efforts and the design of a vaccination policy to mitigate the effect of the novel coronavirus.

1. Introduction

The number of confirmed cases reported by the World Health Organization (WHO) until July 25, 2024, has moved to 775.7 million, whereas the number of deaths has shot to around 7.054 million (World Health Organization (2024)). The novel coronavirus pandemic and the increase in diabetes mellitus (DM) individuals are of utmost concern worldwide (Liu et al. (2022)). Around 11.3% of the United States population is coping with DM (CDC NDSS (2020)). For individuals undergoing treatment for novel coronavirus, DM is the most common comorbidity (Muniyappa and Gubbi (2020)). Increasing indications have entrenched the connection between DM and raised risk of severity of COVID-19 infection and death (Muniyappa and Gubbi (2020); Barron et al., 2020; McGurnaghan et al., 2021; Rawshani et al., 2021). People

with type 2 DM had 1.4 times more tendency to hospital and intensive care unit (ICU) admittance. Additionally, they had a 1.5-fold increased risk of mortality (Rawshani et al. (2021)). Due to the epidemic, interruptions in medical care, being at home, and lagging in the clinical process have led to a decline in the testing process and an increase in the online consultation and medication by persons with diabetes (Patel et al. (2021)). Thus, an increase in uncontrolled glycemic and incidents of diabetic ketoacidosis have been reported (Czeisler et al. (2021)).

Like other viruses, coronavirus also evolves with time. Most modifications have little to no impact on the virus's properties. Nonetheless, a few of these modifications might impact the virus's properties, including how quickly it propagates and the intensity and severity of the illness it causes (Özköse et al. (2022)). The novel coronavirus's omicron variant (B.1.1.529) was initially found in South Africa at the end of November

Abbreviations: COVID-19, Coronavirus Disease 2019; ICU, Intensive Care Unit; DM, Diabetes Mellitus; OV, Omicron Variant; DFE, Disease Free Equilibrium; EE, Endemic Equilibrium; CFR, Case fatality rate.

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2021 (World Health Organization (2021)). After that, it spread globally by the end of January 2022. The omicron variant (OV) was among the more prevalent COVID-19 variants, representing around 85% of total cases (GISAID (2022)). The OV soon replaced the present delta variant in Japan and reached around 94% of total cases reported on January 10–16, 2022 (N.I. of Infectious Diseases (2022)). Contrary to the widespread of OV (Ogata et al. (2022); Jørgensen et al., 2022), numerous studies reported that the severity attached to the delta variant is more than OV (Nyberg et al. (1033); Auvigne et al., 2022; Abdullah et al., 2022; Wang et al., 2023; Esper et al., 2023; Sheikh et al., 2022; Xue et al., 2022). On the other hand, the severity of the novel coronavirus was not just the consequence of the variants. Still, it was influenced by many factors like vaccination (Holman et al. (2020); Gram et al., 2022; Agrawal et al., 2022; McConeghy et al., 2022; Smith et al., 2022), medical facilities, accessibility of coronavirus drugs, and the life expectancy of people before the coronavirus pandemic. The occurrence of OV (BA.1) sublineage was passed by the OV (BA.2) in April, which was overtaken by OV (BA.5) at the end of July and became accountable for around 97% of total reported cases between 15 and 21 August 2022 (N.I. of Infectious Diseases (2022)). The pattern of severity of cases with the omicron variant may have been altered by these differences in viral sublineage penetration, which affected the case fatality rate (CFR). CFR influences novel coronavirus policy-making. The comparison between the severity of OV (BA.4/BA.5) and OV (BA.1) was conducted, and it concluded that a similar risk of hospitalization and death was noticed for both the OV (Davies et al. (2022)). Despite this, there is little information about the CFR's actual temporal changes with distinct OV in Japan (Takahashi et al. (2023)).

The common comorbidity found in patients hospitalized due to novel coronavirus was DM (Zhou et al. (2020)). Most of the available information suggested that the people suffering from DM were at serious risk of hospitalization and death disproportionately affected by severe novel coronavirus (Barron et al. (2020); Singh et al., 2020). People from any age group with conditions such as chronic kidney disease, chronic obstructive pulmonary disease, heart disease, coronary artery disease, type 2 diabetes, and hypertension were at significant risk of severe coronavirus disease (Centers for Disease Control and Prevention (2019)). DM pervasiveness was higher in people with severity compared to people with mild symptoms of coronavirus (Okoye (2020)). Patients with DM are more prone to several viral infections (Badawi and Ryoo (2016); Carey et al., 2018). A meta-analysis of novel coronavirus showed a 9.7% prevalence for diabetes, approximately equivalent to the overall DM episodes in China (Li et al. (2020)). A study on coronavirus was conducted in the United Kingdom and found that around one-third of 23,804 patients had type 2 diabetes, while only 2% had type 1 diabetes. DM patients have a higher risk of death from COVID-19 by around 2.03–3.5 times compared to non-sufferers (Barron et al. (2020)). Heart-related disease is more likely to occur in individuals who have diabetes and coronavirus disease (Shi et al. (2020)). As per the population-based cohort study from England, persons with diabetes who had HbA1c less than 6.5% were less susceptible to coronavirus compared to those who had glycated hemoglobin of more than 10% (Holman et al. (2020)). Yao et al. (Yao et al. (2023)) highlighted the raised diabetes death cases, spatiotemporal arrangement, and related demographic discrepancies during the epidemic. The study also emphasizes keeping track of the evolution of DM. Lima-Martínez et al. (2021) conducted a review of the bidirectional relationship between novel coronavirus and DM. Individuals suffering from DM have a greater risk of suffering when they contract coronavirus; on the other hand, COVID-19 may constitute a diabetogenic agent binding to the ACE2 in β cells, creating intense dysfunction and disturbing glucose levels. In novel coronavirus-infected population under 60 years of age, a single genetic risk factor (3p21.31 locus) may forecast mortality, heart failure, and several known comorbidities like type 2 DM (Niemi et al. (2022)). Till today, there is no precise data on impact of novel coronavirus epidemic on persistent complications related to DM. Nevertheless, optimizing DM management

to slow down the increasing burden on the healthcare system is still essential. Landstra et al. (2021) also explained the bidirectional relationship between DM and COVID-19. According to this work, DM is one of the most paramount risk factors for a severe novel coronavirus case. Besides, a serious coronavirus infection and steroid treatment may increase insulin resistance and β -cells dysfunction. The challenges still exist in understanding the relationship between DM and coronavirus disease.

Therefore, numerous mathematical studies have been conducted to study infectious diseases. These studies explored various aspects of the COVID-19 pandemic and its relationship with comorbidities. Oname et al. (2022) developed a model to study the coronavirus co-dynamics with diabetes and concluded that to control diabetes and novel coronavirus comorbidities, policies must be planned for mass vaccination and strict face-mask usage in public places. Due to the mutation of the virus involved in COVID-19, different variants continue to grow and transmit across the globe. It becomes essential to study the impact of COVID-19 on other comorbidities, particularly people with high-risk diseases such as diabetes. Das et al. (2021) developed a mathematical model to understand the interrelationship between coronavirus and comorbidities. It concluded that novel coronavirus prevailed with the rise in exposed cases with comorbidities. Ssebuliba et al. (2022) developed a deterministic mathematical model to characterize the transmission of novel coronavirus with comorbidities. The size-dependent area was considered in this work to incorporate the effect of social distancing. It is wrapped up with the observation that community transmission increases if the detection rate decreases for the population with and without comorbidities. Oname et al. (2021) formulated a model for transmitting coronavirus dynamics and determined the impact of comorbidities on novel coronavirus complications. Anusha and Athithan (2021) developed a mathematical model for diabetes and a novel coronavirus. This study concluded that the severity of coronavirus cases rises with comorbidities. The spread of COVID-19 disease is much higher in individuals with DM in comparison to the normal population. Liu et al. (2023) studied the persuasiveness of the coronavirus vaccine against death due to any cause in individuals with type 2 DM. The study concluded that the COVID-19 vaccine may prevent death in patients with DM during the omicron variant outbreak. The effect of a new coronavirus on individuals with diabetes and healthy individuals was investigated by Anand et al. (2023). Kouidere et al. (2021) emphasized the negative effect of quarantine on individuals with diabetes. Özköse et al. (2022) studied the interplay between diabetes and COVID-19 through actual data. Kumari et al. (2022) investigated environmental variables affect the prevalence of novel coronavirus. Khan et al. (2021) developed a discrete-time model for individuals with diabetes and exposed to the novel coronavirus. The role of vaccination in preventing the spread of new coronaviruses is recognized. Different computational frameworks have been constructed to examine the effect of vaccination (Kavya et al. (2024); Theparod et al., 2023).

Mathematical modeling is a robust technique for creating a theoretical framework that helps in analyzing real-world situations (Bekiros et al. (2020); Rao and Kumar, 2015; Tyagi et al., 2021; Kumari et al., 2023). A mathematical model can present complex situations on paper in cases where experimenting is complex and costly. Moreover, mathematical models offer advancement and alteration. One can revisit the model to make it close to a real-world scenario. Motivated by the above development, this work concentrates on developing a generalized mathematical model to understand the impact of COVID-19 on the diabetic population. This model considered compartments such as infectious with COVID-19 and diabetes, hospitalization, death, and omicron variant COVID-19. In addition, we considered the effect of vaccination doses, including booster dose. This paper is arranged as follows. Section 2, represents the formulation of mathematical model considering the paramount compartments such as quarantine, hospitalization, COVID-19 with omicron variants, COVID-19 and diabetes infectious, vaccination, and deaths. The boundedness of the solutions for

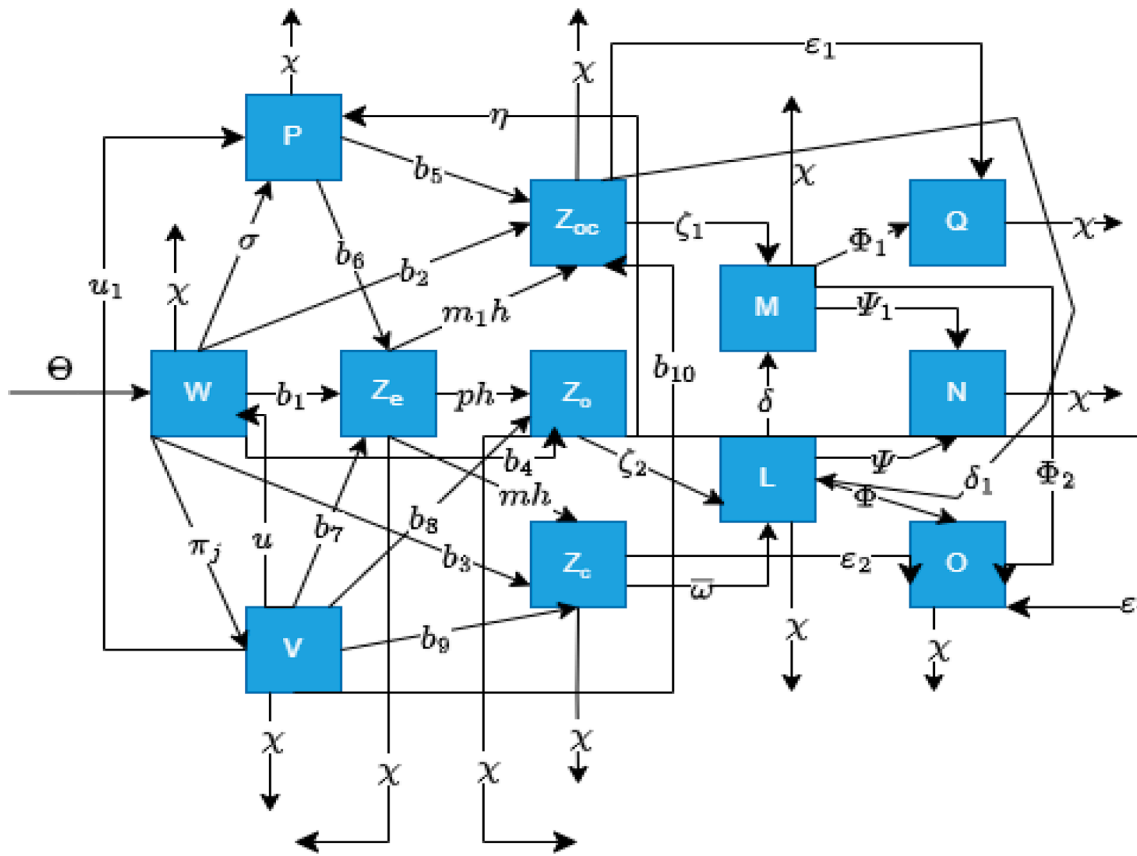


Fig. 1. Compartmental diagram for comorbidities disease dynamics.

the proposed model, computation of reproduction number (R_0), and existence and stability of equilibria are conferred in Section 3. Section 4 deals with numerical simulation and results. In the end, the discussion and conclusion are presented in Section 5.

2. Mathematical model

The statistical trends show that novel coronavirus patients with diabetes are more likely to be admitted to a hospital in a critical care unit (Rawshani et al. (2021)). A comprehensive mathematical model must be considered to capture the dynamics of novel coronavirus in detail. Such a model adds characteristics such as co-infection, vaccinations, hospitalization, multiple variants, and comorbidities. Thus, to visualize the scenario, we develop a new mathematical model for the co-infection of diabetes and novel coronavirus patients. This model consists of twelve compartments such as susceptible (W), exposed (Z_e), novel coronavirus infectious omicron variant (Z_o), COVID-19 infectious other than omicron variant (Z_c), infected with co-infection (Z_{oc}), quarantined (L), hospitalized (M), recovered (N), deaths (O), diabetes (P), death due to co-infection (Q), and vaccinated population (V). The relationship between different populations is illustrated in Fig. 1. The total population $\Xi = W + V + P + Z_e + Z_c + Z_{oc} + Z_o + L + M + N + O + Q$ is assumed to be constant. From a biological perspective, the proposed nonlinear mathematical model for diabetes and novel coronavirus is represented by the following equations.

$$\begin{aligned}
 \frac{dW(\tau)}{d\tau} &= \Theta - (b_1 + b_2 + b_3 + b_4)W(\tau) + uV(\tau) - (\sigma + \chi + \pi_j)W(\tau), \\
 \frac{dV(\tau)}{d\tau} &= \pi_j W(\tau) - (b_7 + b_8 + b_9 + b_{10})V(\tau) - (u + u_1 + \chi)V(\tau), \\
 \frac{dP(\tau)}{d\tau} &= \sigma W(\tau) + \eta N(\tau) + u_1 V(\tau) - (b_5 + b_6)P(\tau) - \chi P(\tau), \\
 \frac{dZ_e(\tau)}{d\tau} &= b_1 W(\tau) + b_6 P(\tau) + b_7 V(\tau) - (h + \chi)Z_e(\tau), \\
 \frac{dZ_c(\tau)}{d\tau} &= b_3 W(\tau) + b_9 V(\tau) + m_1 h Z_e(\tau) - (\varpi + \epsilon_2 + \chi)Z_c(\tau), \\
 \frac{dZ_{oc}(\tau)}{d\tau} &= b_2 W(\tau) + b_5 P(\tau) + b_{10} V(\tau) + m h Z_e(\tau) - (\zeta_1 + \delta_1 + \epsilon_1 + \chi)Z_{oc}(\tau), \\
 \frac{dZ_o(\tau)}{d\tau} &= b_4 W(\tau) + b_8 V(\tau) + p h Z_e(\tau) - (\zeta_2 + \epsilon + \chi)Z_o(\tau), \\
 \frac{dL(\tau)}{d\tau} &= \varpi Z_c(\tau) + \zeta_2 Z_o(\tau) + \delta_1 Z_{oc}(\tau) - (\delta + \Psi(\tau) + \Phi(\tau) + \chi)L(\tau), \\
 \frac{dM(\tau)}{d\tau} &= \zeta_1 Z_{oc}(\tau) + \delta L(\tau) - (\Psi_1(\tau) + \Phi_1(\tau) + \Phi_2(\tau) + \chi)M(\tau), \\
 \frac{dN(\tau)}{d\tau} &= \Psi(\tau)L(\tau) + \Psi_1(\tau)M(\tau) - (\eta + \chi)N(\tau), \\
 \frac{dO(\tau)}{d\tau} &= \Phi(\tau)L(\tau) + \Phi_2(\tau)M(\tau) + \epsilon_2 Z_c(\tau) + \epsilon Z_o(\tau) - \chi O(\tau), \\
 \frac{dQ(\tau)}{d\tau} &= \Phi_1(\tau)M(\tau) + \epsilon_1 Z_{oc}(\tau) - \chi Q(\tau),
 \end{aligned} \tag{2.1}$$

Table 1
Symbols and their definition.

Symbol	Definition
Θ	Requirement rate
ϖ, ζ_2	Quarantine rate for variants other than omicron and omicron variant
ϑ	Infection rate
ϑ_1, ϑ_3	Infection rate for exposed without comorbidities and co-infection
ϵ_1, ϵ_2	Disease-induced death rate for omicron and variants other than omicron
ϵ_1	Disease-induced death rate for co-infection
δ_1	Quarantine rate for co-infection
π_j	Vaccination rate
$\Phi_2(\tau)$	Mortality rate from hospitalized to population without comorbidities (Cheynet (2020))
η_j	Vaccine efficacy
u	Vaccine ineffectiveness rate
u_1	Vaccine ineffectiveness rate for diabetic
h	Inverse of average latent time
σ	Rate of diabetes individuals
Ξ	Total Population
ϑ_2	Infection rate (co-infection)
η	Rate of shift from N to P compartment
m	Probability for co-infection
m_1	Probability for COVID-19 population for variant other than omicron
χ	Natural death rate
$\Psi(\tau), \Psi_1(\tau)$	Recovery rate for COVID-19 and co-infection (Cheynet (2020))
$\Phi(\tau), \Phi_1(\tau)$	Mortality rate for COVID-19 and co-infection (Cheynet (2020))
ζ_1	Hospitalized rate for co-infection

with initial condition $X = \{W(0) = W_0 > 0, V(0) = V_0 \geq 0, P(0) = P_0 \geq 0, Z_e(0) = Z_{e_0} \geq 0, Z_c(0) = Z_{c_0} \geq 0, Z_o(0) = Z_{o_0} \geq 0, Z_{oc}(0) = Z_{oc_0} \geq 0, L(0) = L_0 \geq 0, M(0) = M_0 \geq 0, N(0) = N_0 \geq 0, O(0) = O_0 \geq 0, Q(0) = Q_0 \geq 0\}$,

(2.2)

and

$$p = \left(1 - m - m_1\right), b_1 = \frac{\vartheta_1 Z_e}{\Xi}, b_2 = \frac{\vartheta Z_{oc}}{\Xi}, b_3 = \frac{\vartheta Z_c}{\Xi}, b_4 = \frac{\vartheta Z_o}{\Xi},$$

$$b_5 = \frac{\vartheta(Z_c + Z_o)}{\Xi}, b_6 = \frac{\vartheta_3 Z_e}{\Xi}, b_7 = \frac{\vartheta_1(1 - \eta_j)Z_e}{\Xi}, b_8 = \frac{\vartheta(1 - \eta_j)Z_o}{\Xi},$$

$$b_9 = \frac{\vartheta(1 - \eta_j)Z_c}{\Xi}, b_{10} = \frac{\vartheta(1 - \eta_j)Z_{oc}}{\Xi}.$$

The parameters for model (2.1) are defined in Table 1. Because of their biological significance, all parameters must be non-negative. Since generally no symptoms appear during the latent period, we consider individuals in the exposed compartment to be in the asymptomatic phase. When symptoms do appear after the latent period, the population is considered in the symptomatic stage of the infection. The transmission of novel coronavirus is possible through symptomatic and asymptomatic infectious (Bhadauria et al. (2022)). We consider the spread of disease in susceptible compartment through infectious and exposed compartments at the rate ϑ and ϑ_1 , respectively. The vaccinated population is less prone to the disease than people without vaccination. The non-vaccinated susceptible individuals who come in contact with asymptomatic infected individuals also get exposed to COVID-19 with transmission coefficient ϑ_1 . The susceptible individuals with dose-1, dose-2, and booster dose contacted with asymptomatic infectious get exposed to COVID-19 with transmission coefficient $(1 - n_1)\vartheta_1, (1 - n_2)\vartheta_1, (1 - n_3)\vartheta_1$, respectively. The non-vaccinated susceptible individuals who come in contact with symptomatic omicron-infected individuals get exposed to COVID-19 with transmission coefficient ϑ . The susceptible dose-1, dose-2, and

booster dose population contacted with symptomatic infected individuals with omicron get exposed to COVID-19 with a reduced rate $(1 - n_1)\vartheta, (1 - n_2)\vartheta, (1 - n_3)\vartheta$, respectively. The following assumptions are considered for the proposed model.

- Natural recruitment is thought to be responsible for the increase in population (Bichara et al. (2014)).
- All compartments account for natural death rates (Bichara et al. (2014)).
- Both individuals with and without symptoms can spread the virus (Nogady (2020)).
- Due to vaccine inefficiency, some previously vaccinated persons may become vulnerable again (Negi et al. (2022); Rana et al., (2022)).
- Because reinfections account for a small percentage of total infectious, we assume that immunity established after recovery is long-term (Massard et al. (2022)).
- Diabetes is a medical condition resulting from an abnormal glucose level rise. Therefore, it cannot be transmitted between individuals (Anusha and Athithan (2021)).
- The population without diabetes can remain in the recovered compartment, but individuals with diabetes are sent back to the diabetes compartment (Anusha and Athithan (2021)).

3. Analytical Analysis

This section is devoted to analytical investigations of the proposed model (2.1). We ascertain the invariant region and the boundedness requirement. In addition, stability analysis, computation of equilibrium points, and reproduction number R_0 are carried out.

$$\text{Let } \mathbb{R}_+^{12} = \{\theta(\tau) \in \mathbb{R}^{12} : \theta(\tau) \geq 0\} \text{ and}$$

$$\theta(\tau) = [W(\tau), V(\tau), P(\tau), Z_e(\tau), Z_c(\tau), Z_{oc}(\tau), Z_o(\tau), L(\tau), M(\tau), N(\tau), O(\tau), Q(\tau)]^T. \quad (3.1)$$

Lemma 3.1. Assume that $g(x) \in C[c, d]$ and $g'(x) \in C[c, d]$. It follows from the Mean Value Theorem that if $g'(x) \geq 0, \forall x \in (c, d)$ then $g(x)$ is non-decreasing $\forall x \in (c, d)$.

Theorem 3.2. The solutions to model (2.1) under initial condition (2.2) exhibit positive invariance.

Proof. It needs to be determined that region $\mathbb{R}_+^{12}$ is positively invariant. From system (2.1), we have

$$\left. \frac{dW(\tau)}{d\tau} \right|_{W=0} = \Theta + uV(\tau) > 0,$$

$$\left. \frac{dV(\tau)}{d\tau} \right|_{V=0} = \pi_j W(\tau) \geq 0,$$

$$\left. \frac{dP(\tau)}{d\tau} \right|_{P=0} = \sigma W(\tau) + \eta N(\tau) + u_1 V(\tau) \geq 0,$$

$$\left. \frac{dZ_e(\tau)}{d\tau} \right|_{Z_e=0} = b_1 W(\tau) + b_6 P(\tau) + b_7 V(\tau) \geq 0,$$

$$\left. \frac{dZ_c(\tau)}{d\tau} \right|_{Z_c=0} = b_3 W(\tau) + b_9 V(\tau) + m_1 h Z_e(\tau) \geq 0,$$

$$\left. \frac{dZ_{oc}(\tau)}{d\tau} \right|_{Z_{oc}=0} = b_2 W(\tau) + b_5 P(\tau) + b_{10} V(\tau) + m h Z_e(\tau) \geq 0,$$

$$\begin{aligned} \left. \frac{dZ_o(\tau)}{d\tau} \right|_{Z_o=0} &= b_4 W(\tau) + b_8 V(\tau) + phZ_e(\tau) \geq 0, \\ \left. \frac{dL(\tau)}{d\tau} \right|_{L=0} &= \varpi Z_c(\tau) + \zeta_2 Z_o(\tau) + \delta_1 Z_{oc}(\tau) \geq 0, \\ \left. \frac{dM(\tau)}{d\tau} \right|_{M=0} &= \zeta_1 Z_{oc}(\tau) + \delta L(\tau) \geq 0, \\ \left. \frac{dN(\tau)}{d\tau} \right|_{N=0} &= \Psi(\tau)L(\tau) + \Psi_1(\tau)M(\tau) \geq 0, \\ \left. \frac{dO(\tau)}{d\tau} \right|_{O=0} &= \Phi(\tau)L(\tau) + \Phi_2(\tau)M(\tau) + \epsilon_2 Z_c(\tau) + \epsilon Z_o(\tau) \geq 0 \\ \left. \frac{dQ(\tau)}{d\tau} \right|_{Q=0} &= \Phi_1(\tau)M(\tau) + \epsilon_1 Z_{oc}(\tau) \geq 0. \end{aligned}$$

If $(W_0, V_0, P_0, Z_{e0}, Z_{c0}, Z_{oc0}, Z_{o0}, L_0, M_0, N_0, O_0, Q_0) \in \mathbb{R}_+^{12}$ then in accord with equations in (3.2) and (Lemma 3.1), solutions of model (2.1) be unable to escape the hyperplane $W = 0, V = 0, P = 0, Z_e = 0, Z_c = 0, Z_o = 0, Z_{oc} = 0, L = 0, M = 0, N = 0, O = 0, Q = 0$. Therefore, the area $\mathbb{R}_+^{12}$ is positively invariant set.

$$\begin{aligned} \Theta - (b_1 + b_2 + b_3 + b_4)W(\tau) + uV(\tau) - (\sigma + \pi_j + \chi)W(\tau) &= 0, \\ \pi_j W(\tau) - (b_7 + b_8 + b_9 + b_{10})V(\tau) - (u + u_1 + \chi)V(\tau) &= 0, \\ \sigma W(\tau) + \eta N(\tau) + u_1 V(\tau) - (b_5 + b_6)P(\tau) - \chi P(\tau) &= 0, \\ b_1 W(\tau) + b_6 P(\tau) + b_7 V(\tau) - (h + \chi)Z_e(\tau) &= 0, \\ b_3 W(\tau) + b_9 V(\tau) + m_1 h Z_e(\tau) - (\varpi + \epsilon_2 + \chi)Z_c(\tau) &= 0, \\ b_2 W(\tau) + b_5 P(\tau) + b_{10} V(\tau) + m h Z_e(\tau) - (\zeta_1 + \delta_1 + \epsilon_1 + \chi)Z_{oc}(\tau) &= 0, \\ b_4 W(\tau) + b_8 V(\tau) + phZ_e(\tau) - (\zeta_2 + \epsilon + \chi)Z_o(\tau) &= 0, \\ \varpi Z_c(\tau) + \zeta_2 Z_o(\tau) + \delta_1 Z_{oc}(\tau) - (\delta + \Psi(\tau) + \Phi(\tau) + \chi)L(\tau) &= 0, \\ \zeta_1 Z_{oc}(\tau) + \delta L(\tau) - (\Psi_1(\tau) + \Phi_1(\tau) + \Phi_2(\tau) + \chi)M(\tau) &= 0, \\ \Psi(\tau)L(\tau) + \Psi_1(\tau)M(\tau) + \Phi_2(\tau)M(\tau) - (\eta + \chi)N(\tau) &= 0, \\ \Phi(\tau)L(\tau) + \epsilon_2 Z_c(\tau) + \epsilon Z_o(\tau) - \chi O(\tau) &= 0, \\ \Phi_1(\tau)M(\tau) + \epsilon_1 Z_{oc}(\tau) - \chi Q(\tau) &= 0. \end{aligned} \quad (3.4)$$

The DFE point for system (2.1) is $D_0 = (W_d, V_d, P_d, 0, 0, 0, 0, 0, 0, 0, 0, 0)$, where

$$W_d = \frac{\Theta(u+u_1+\chi)}{(\sigma+\chi)(u+u_1+\chi)+\pi_j(u_1+\chi)}, \quad V_d = \frac{\Theta\pi_j}{(u+u_1+\chi)(\sigma+\chi)+\pi_j(u_1+\chi)}, \quad P_d = \frac{\sigma\Theta(u+u_1+\chi)+u_1\Theta\pi_j}{\chi((\sigma+\chi)(u+u_1+\chi)+\pi_j(u_1+\chi))}.$$

Now to find the endemic equilibrium (EE), let $(W^*, V^*, P^*, Z_e^*, Z_c^*, Z_{oc}^*, Z_o^*, L^*, M^*, N^*, O^*, Q^*)$ be EE point, we can get it by solving system (3.4)

$$\left\{ \begin{aligned} V^* &= \frac{-\Theta + (b_7 + b_8 + b_9 + b_{10})W^* + (\sigma + \pi_j + \chi)W^*}{u}, & Z_e^* &= \frac{b_1 W^* + b_6 P^* + b_7 V^*}{h + \chi}, \\ Z_c^* &= \frac{b_3 W^* + b_9 V^* + m_1 h Z_e^*}{(\varpi + \epsilon_2 + \chi)}, & Z_{oc}^* &= \frac{b_2 W^* + b_{10} V^* + b_5 P^* + m h Z_e^*}{(\zeta_1 + \delta_1 + \epsilon_1 + \chi)}, \\ Z_o^* &= \frac{b_4 W^* + b_8 V^* + phZ_e^*}{(\zeta_2 + \epsilon + \chi)}, & L^* &= \frac{(\zeta)Z_c^* + \zeta_2 Z_o^* + \delta_1 Z_{oc}^*}{(\Psi + \Phi + \delta + \chi)}, \\ M^* &= \frac{\zeta_1 Z_{oc}^* + \delta L^*}{(\Psi_1 + \Phi_1 + \Phi_2 + \chi)}, & N^* &= \frac{\Psi L^* + \Psi_1 M^*}{\eta + \chi}, \\ O^* &= \frac{\Phi L^* + \Phi_2 M^* + \epsilon Z_o^* + \epsilon_2 Z_c^*}{\chi}, & Q^* &= \frac{M^* \Phi_1}{\chi}. \end{aligned} \right. \quad (3.5)$$

Theorem 3.3. The region Ω acts as a positively invariant set for model (2.1), where

$$\Omega = \{(W(\tau), V(\tau), P(\tau), Z_e(\tau), Z_c(\tau), Z_{oc}(\tau), Z_o(\tau), L(\tau), M(\tau), N(\tau), O(\tau), Q(\tau)) \in \mathbb{R}_+^{12} : 0 < \Xi \leq \frac{\Theta}{\chi}\}.$$

Proof. From system (2.1), we get

$$\begin{aligned} \frac{d\Xi}{d\tau} &= \Theta - \chi(W(\tau) + V(\tau) + P(\tau) + Z_e(\tau) + Z_c(\tau) + Z_{oc}(\tau) + Z_o(\tau) + L(\tau) + M(\tau) + N(\tau) + O(\tau) + Q(\tau)) \text{ which implies} \\ \frac{d\Xi}{d\tau} + \chi \Xi &\leq \Theta \end{aligned} \quad (3.3)$$

Now solving Eq. (3.3), we get

$$\Xi \leq \frac{\Theta}{\chi} + \left(\Xi(\tau_1) - \frac{\Theta}{\chi} \right) e^{-\chi\tau}, \quad \forall \tau \in (0, \infty) \text{ for } \tau \rightarrow \infty, \left(\Xi(\tau_1) - \frac{\Theta}{\chi} \right) e^{-\chi\tau} \rightarrow 0,$$

which implies $0 < \Xi \leq \frac{\Theta}{\chi}$.

Hence, the solutions to the system (2.1) reside in $\mathbb{R}_+^{12}$ establishing a biologically plausible region in Ω .

3.1. Computation of equilibrium

A situation in which COVID-19 is eradicated from the population is a known as disease-free equilibrium (DFE) point. To determine DFE point of system (2.1), we solve system (3.4) and assume the state variables $Z_e, Z_c, Z_{oc},$ and Z_o to be zero.

3.2. Basic reproduction number (R_0)

To find the value of R_0 , we utilize the next-generation matrix (NGM) method (Diekmann et al. (2010); El-Sayed et al., 2007). The compartments $Z_e, Z_c, Z_{oc},$ and Z_o in model (2.1) are specifically included with disease transmission. The transmission and transition matrices are presented by

$$\mathcal{F} = \begin{bmatrix} \frac{\partial_1 Z_e W_d}{\Xi} + \frac{\partial_3 Z_e P_d}{\Xi} + \frac{(1 - n_j) \partial_1 Z_e V_d}{\Xi} \\ \frac{\partial Z_c W_d}{\Xi} + \frac{(1 - n_j) \partial Z_c V_d}{\Xi} \\ \frac{\partial Z_{oc} W_d}{\Xi} + \frac{\partial_2 (Z_c + Z_o) P_d}{\Xi} + \frac{(1 - n_j) \partial Z_{oc} V_d}{\Xi} \\ \frac{\partial Z_o W_d}{\Xi} + \frac{(1 - n_j) \partial Z_o V_d}{\Xi} \end{bmatrix} \quad \text{and} \quad \mathcal{V} = \begin{bmatrix} -(h + \chi) Z_e \\ m_1 h Z_e - (\varpi + \epsilon_2 + \chi) Z_c \\ m h Z_e - (\zeta_1 + \epsilon_1 + \delta_1 + \chi) Z_{oc} \\ ph Z_e - (\zeta_2 + \epsilon + \chi) Z_o \end{bmatrix},$$

respectively.

The Jacobian matrices for $\mathcal{F}$ and $\mathcal{V}$ at the DFE point are provided as

$$F = \begin{bmatrix} \frac{\partial_1 W_d}{\Xi} + \frac{\partial_3 P_d}{\Xi} + \frac{(1-n_j)\partial_1 V_d}{\Xi} & 0 & 0 & 0 \\ 0 & \frac{\partial W_d}{\Xi} + \frac{(1-n_j)\partial V_d}{\Xi} & 0 & 0 \\ 0 & \frac{\partial_2 P_d}{\Xi} & \frac{\partial W_d}{\Xi} + \frac{(1-n_j)\partial V_d}{\Xi} & \frac{\partial_2 P_d}{\Xi} \\ 0 & 0 & 0 & \frac{\partial W_d}{\Xi} + \frac{(1-n_j)\partial V_d}{\Xi} \end{bmatrix} \text{ and}$$

$$\mathcal{V} = \begin{bmatrix} -(h+\chi) & 0 & 0 & 0 \\ m_1 h & -(\varpi + \epsilon_2 + \chi) & 0 & 0 \\ m h & 0 & -(\zeta_1 + \epsilon_1 + \delta_1 + \chi) & 0 \\ (1-m-m_1)h & 0 & 0 & -(\zeta_2 + \epsilon + \chi) \end{bmatrix}, \text{ respectively.}$$

Computing the spectral radius of the matrix $F\mathcal{V}^{-1}$, the expression for $R_0 = \max\{R_1, R_2, R_3, R_4\}$, where $R_1 = \frac{\partial V_d(1-n_j)+\partial W_d}{\Xi(\epsilon_2+\chi+\varpi)}$, $R_2 = \frac{\partial V_d(1-n_j)+\partial W_d}{\Xi(\zeta_2+\chi+\epsilon)}$, $R_3 = \frac{\partial V_d(1-n_j)+\partial W_d}{\Xi(\chi+\delta_1+\epsilon_1+\zeta_1)}$, $R_4 = \frac{\partial V_d(1-n_j)+\partial W_d+\partial_3 P_d}{\Xi(\chi+h)}$.

3.3. Stability analysis

This subsection deals with local and global stability analysis of DFE point.

Theorem 3.4. If $R_0 < 1$, then the DFE point of system (2.1) is locally asymptotically stable.

Proof. To verify local stability of DFE, the Jacobian matrix for system (2.1) at D_0 presented as follows

$$K_{D_0} = \begin{bmatrix} -(\chi + \sigma + \pi_j) & u & 0 & \frac{\partial_1 W_d}{\Xi} & \frac{\partial W_d}{\Xi} & \frac{\partial W_d}{\Xi} & \frac{\partial W_d}{\Xi} & 0 & 0 & 0 & 0 & 0 \\ \pi_j & -(u + u_1 + \chi) & 0 & \frac{\partial_1(1-n_j)V_d}{\Xi} & \frac{\partial(1-n_j)V_d}{\Xi} & \frac{\partial(1-n_j)V_d}{\Xi} & \frac{\partial(1-n_j)V_d}{\Xi} & 0 & 0 & 0 & 0 & 0 \\ \sigma & u_1 & -\chi & \frac{\partial_3 P_d}{\Xi} & \frac{\partial_2 P_d}{\Xi} & 0 & \frac{\partial_2 P_d}{\Xi} & 0 & 0 & \eta & 0 & 0 \\ 0 & 0 & 0 & a - (h + \chi) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & h m_1 & b - (\chi + \varpi + \epsilon_2) & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & h m & \frac{\partial_2 P_d}{\Xi} & b - (\chi + \zeta_1 + \delta_1 + \epsilon_1) & \frac{\partial_2 P_d}{\Xi} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & h(1-m-m_1) & 0 & 0 & b - (\chi + \zeta_2 + \epsilon) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \zeta & \delta_1 & \zeta_2 & -d & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \zeta_1 & 0 & \delta & -c & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \psi & \psi_1 & -(\chi + \eta) & 0 & 0 \\ 0 & 0 & 0 & 0 & \epsilon_2 & 0 & \epsilon & \Phi & \Phi_2 & 0 & -\chi & 0 \\ 0 & 0 & 0 & 0 & 0 & \epsilon_1 & 0 & 0 & \Phi_1 & 0 & 0 & -\chi \end{bmatrix}$$

where, $a = \frac{\partial_3 P_d}{\Xi} + \frac{\partial_2((1-n_j)V_d + W_d)}{\Xi}$, $b = \frac{\partial((1-n_j)V_d + W_d)}{\Xi}$, $c = (\chi + \Phi_1 + \Phi_2 + \psi_1)$, and $d = (\delta + \chi + \Phi + \psi)$.

The Jacobian matrix K_{D_0} has eigenvalues.

$$\begin{aligned} k_{1,2,3} &= -\chi, -\chi, -\chi, k_4 = -\chi - \eta, k_5 = -c, k_6 = -d, k_7 \\ &= -(u + \sigma + \chi + \pi_j), k_8 = -(u_1 + \chi), k_9 = -(1 - R_4)(h + \chi), k_{10} \\ &= -(\chi + \zeta_1 + \delta_1 + \epsilon_1)(1 - R_3), k_{11} = -(1 - R_2)(\chi + \zeta_2 + \epsilon), \text{ and } k_{12} \\ &= -(1 - R_1)(\chi + \varpi + \epsilon_2). \end{aligned}$$

Since all the eigenvalues of K_{D_0} have negative real part if $R_0 < 1$. Thus, model (2.1) is locally asymptotically stable at D_0 . Furthermore, the following theorem proves that the DFE point is globally asymptotically stable (GAS).

Theorem 3.5. The DFE point D_0 of the system (2.1) is GAS if $R_0 < 1$ and conditions in a) and b) are true:

a) $\frac{dH}{dt} = \mathcal{F}(H, 0)$, H_0 is globally asymptotically stable.

b) $\tilde{\Omega}(H, \Delta) \geq 0$ for $(H, \Delta) \in \mathfrak{F}$, where $\tilde{\Omega}(H, \Delta) = \mathfrak{G}\Delta - \Omega(H, \Delta)$, $\mathfrak{G} = D_\Delta \Omega(H_0, 0)$ is M -matrix and $\mathfrak{F} = \{(W(\tau), Z_e(\tau), Z_c(\tau), Z_{oc}(\tau), Z_o(\tau), L(\tau), M(\tau), N(\tau), O(\tau), P(\tau), Q(\tau), V(\tau)) \in \mathbb{R}_+^{12} : 0 < \Xi \leq \frac{\Theta}{\chi}\}$ is biologically plausible region.

Proof. Employing Feng et al.'s theorem (Castillo-Chavez et al. (2002)), we establish that D_0 is GAS for system (2.1). We first rewrite system (2.1) as follows

$$\frac{dH}{d\tau} = \mathcal{S}(H, \Delta) = \begin{bmatrix} \Theta - (b_1 + b_2 + b_3 + b_4)W(\tau) + uV(\tau) - (\sigma + \pi_j + \chi)W(\tau) \\ \pi_j W(\tau) - (b_7 + b_8 + b_9 + b_{10})V(\tau) - (u + u_1 + \chi)V(\tau) \\ \sigma W(\tau) + \eta N(\tau) + u_1 V(\tau) - (b_5 + b_6)P(\tau) - \chi P(\tau) \\ \varpi Z_c(\tau) + \zeta_2 Z_o(\tau) + \delta_1 Z_{oc}(\tau) - (\delta + \Psi(\tau) + \Phi(\tau) + \chi)L(\tau) \\ \zeta_1 Z_{oc}(\tau) + \delta L(\tau) - (\Psi_1(\tau) + \Phi_1(\tau) + \Phi_2(\tau) + \chi)M(\tau) \\ \Psi(\tau)L(\tau) + \Psi_1(\tau)M(\tau) - (\eta + \chi)N(\tau) \\ \Phi(\tau)L(\tau) + \Phi_2(\tau)M(\tau) + \epsilon_2 Z_c + \epsilon Z_o - \chi O(\tau) \\ \Phi_1(\tau)M(\tau) + \epsilon_1 Z_{oc} - \chi Q(\tau) \end{bmatrix},$$

$$\frac{d\Delta}{d\tau} = \Omega(H, \Delta),$$

$$\Omega(H, \Delta) = \begin{bmatrix} b_1 W(\tau) + b_6 P(\tau) + b_7 V(\tau) - (h + \chi)Z_e(\tau) \\ b_3 W(\tau) + b_9 V(\tau) + m_1 h Z_e(\tau) - (\varpi + \epsilon_2 + \chi)Z_c(\tau) \\ b_2 W(\tau) + b_5 P(\tau) + b_{10} V(\tau) + m h Z_e(\tau) - (\zeta_1 + \delta_1 + \epsilon_1 + \chi)Z_{oc}(\tau) \\ b_4 W(\tau) + b_8 V(\tau) + p h Z_e(\tau) - (\zeta_2 + \epsilon + \chi)Z_o(\tau) \end{bmatrix}, \text{ and}$$

$$\Omega(H, 0) = 0,$$

where $H = (W, V, P, L, M, N, O, Q)$ and $\Delta = (Z_e, Z_c, Z_{oc}, Z_o)$ are non-infectious and infectious compartments, respectively. $\mathcal{S}(H, \Delta)$ and $\Omega(H, \Delta)$, denote (RHS) of system (2.1) corresponding to H and Δ . DFE is reformulated as $D_0 = (H_0, 0, 0, 0, 0)$, with $H_0 = (W_d, V_d, P_d, 0, 0, 0, 0, 0)$.

To show condition a), from system (2.1) we have

$$\mathcal{S}(H, 0) = \begin{bmatrix} \Theta + uV(\tau) - (\sigma + \pi_j + \chi)W(\tau) \\ \pi_j W(\tau) - (u + u_1 + \chi)V(\tau) \\ \sigma W(\tau) + \eta N(\tau) + u_1 V(\tau) - \chi P(\tau) \\ -(\delta + \Psi(\tau) + \Phi(\tau) + \chi)L(\tau) \\ \delta L(\tau) - (\Psi_1(\tau) + \Phi_1(\tau) + \chi)M(\tau) \\ \Psi(\tau)L(\tau) + \Psi_1(\tau)M(\tau) - (\eta + \chi)N(\tau) \\ \Phi(\tau)L(\tau) + \Phi_2(\tau)M(\tau) - \chi O(\tau) \\ \Phi_1(\tau)M(\tau) - \chi Q(\tau) \end{bmatrix}, \quad (3.6)$$

$$W(\tau) = A_1 e^{-\frac{((f+g)-\sqrt{(f-g)^2+4u\pi_j})\tau}{2}} + A_2 e^{-\frac{((f+g)+\sqrt{(f-g)^2+4u\pi_j})\tau}{2}} + W_d,$$

$$V(\tau) = A_3 e^{-\frac{((f+g)-\sqrt{(f-g)^2+4u\pi_j})\tau}{2}} + A_4 e^{-\frac{((f+g)+\sqrt{(f-g)^2+4u\pi_j})\tau}{2}} + V_d,$$

where $f = (\sigma + \pi_j + \chi)$, $g = (u + u_1 + \chi)$, and $A_i, i = 1, 2, 3 \dots$ are arbitrary constants. Similarly, we solve the other equations in system (3.6).

Now, if $\tau \rightarrow \infty$, then $W(\tau) \rightarrow W_d, V(\tau) \rightarrow V_d, P(\tau) \rightarrow P_d$, and $L(\tau), M(\tau), N(\tau), O(\tau)$, and $Q(\tau) \rightarrow 0$.

Therefore, $H \rightarrow H_0$ as $\tau \rightarrow \infty$. Hence, H_0 is globally asymptotically stable and condition a) is satisfied. To show condition b), we consider the RHS of system (2.1) corresponding to Δ and get,

$$\mathcal{G} = \begin{bmatrix} a - (h + \chi) & 0 & 0 & 0 \\ m_1 h & b - (\varpi + \chi + \epsilon_2) & 0 & 0 \\ m h & \frac{\partial_2 P_d}{\Xi} & b - (\zeta_1 + \delta_1 + \epsilon_1 + \chi) & \frac{\partial_2 P_d}{\Xi} \\ (1 - m_1 - m)h & 0 & 0 & b - (\zeta_2 + \epsilon + \chi) \end{bmatrix}$$

solving first and second differential equations from system (3.6), we obtain

putting the value of ($\mathcal{G}$ and $\Omega(H, \Delta)$ in condition b), we obtain

$$\tilde{\Omega}(H, \Delta) = \begin{bmatrix} \frac{\vartheta_3(P_d - P)Z_e}{\Xi} + \frac{\vartheta_2((1 - n_j)(V_d - V) + (W_d - W))Z_e}{\Xi} \\ \frac{Z_c\vartheta((1 - n_j)(V_d - V) + (W_d - W))}{\Xi} \\ \frac{Z_c\vartheta_2(P_d - P)}{\Xi} + \frac{Z_o\vartheta_2(P_d - P)}{\Xi} + \frac{Z_{oc}\vartheta((1 - n_j)(V_d - V) + (W_d - W))}{\Xi} \\ \frac{Z_o\vartheta((1 - n_j)(V_d - V) + (W_d - W))}{\Xi} \end{bmatrix}$$

, which clearly implies that $\tilde{\Omega}(H, \Delta) \geq 0$ for $(H, \Delta) \in \mathfrak{F}$.

3.4. Sensitivity Analysis of R_0

This section addresses sensitivity analysis of parameters that impact R_0 . Sensitivity computation involves assessing how changes in parameters result in changes to R_0 . The sensitivity analysis is determined using the method provided by Chitnis et al. (2008). To ascertain sensitivity, we compute the partial derivative of the variable R_0 concerning various parameters influencing the reproduction number.

$$\begin{aligned} \frac{\partial R_1}{\partial \vartheta} &= \frac{V_d(1 - n_j) + W_d}{(\varpi + \epsilon_2 + \chi)\Xi} > 0, \\ \frac{\partial R_2}{\partial \vartheta} &= \frac{V_d(1 - n_j) + W_d}{(\zeta_2 + \epsilon + \chi)\Xi} > 0, \\ \frac{\partial R_3}{\partial \vartheta} &= \frac{V_d(1 - n_j) + W_d}{(\zeta_1 + \delta_1 + \epsilon_1 + \chi)\Xi} > 0, \\ \frac{\partial R_4}{\partial \vartheta} &= \frac{V_d(1 - n_j) + W_d}{(h + \chi)\Xi} > 0, \\ \frac{\partial R_1}{\partial n_j} &= \frac{-V_d}{(\varpi + \epsilon_2 + \chi)\Xi} < 0, \\ \frac{\partial R_2}{\partial n_j} &= \frac{-V_d}{(\zeta_2 + \epsilon + \chi)\Xi} < 0, \\ \frac{\partial R_3}{\partial n_j} &= \frac{-V_d}{(\zeta_1 + \delta_1 + \epsilon_1 + \chi)\Xi} < 0, \\ \frac{\partial R_4}{\partial n_j} &= \frac{-V_d}{(h + \chi)\Xi} < 0, \\ \frac{\partial R_1}{\partial \varpi} &= \frac{V_d(1 - n_j) + W_d}{(\varpi + \epsilon_2 + \chi)^2\Xi} < 0, \\ \frac{\partial R_2}{\partial \zeta_2} &= \frac{V_d(1 - n_j) + W_d}{(\zeta_2 + \epsilon + \chi)^2\Xi} < 0, \\ \frac{\partial R_3}{\partial \zeta_1} &= \frac{V_d(1 - n_j) + W_d}{(\zeta_1 + \delta_1 + \epsilon_1 + \chi)^2\Xi} < 0, \\ \frac{\partial R_4}{\partial h} &= -\frac{\vartheta(V_d(1 - n_j) + W_d) + \vartheta_3 P_d}{(h + \chi)^2\Xi} < 0, \\ \frac{\partial R_4}{\partial \vartheta_3} &= \frac{P_d}{(h + \chi)\Xi} > 0. \end{aligned} \quad (3.7)$$

From aforementioned analysis, we can infer that R_0 escalates for parameters ϑ, ϑ_3 while diminishes for $\varpi, \zeta_1, \zeta_2, h$. Consequently, to mitigate the pandemic, the parameters with negative partial derivatives should be maximized.

4. Numerical Simulation

In this section, numerical results are conferred to provide more insights into the proposed model. MATLAB software is utilized for

simulations. To find the best-fitted parameters, we use the least square curve fitting technique (MathWorks LSQCURVEFIT (2023)). The parameter values are listed in Table 2. For simulation, the following initial conditions are utilized:

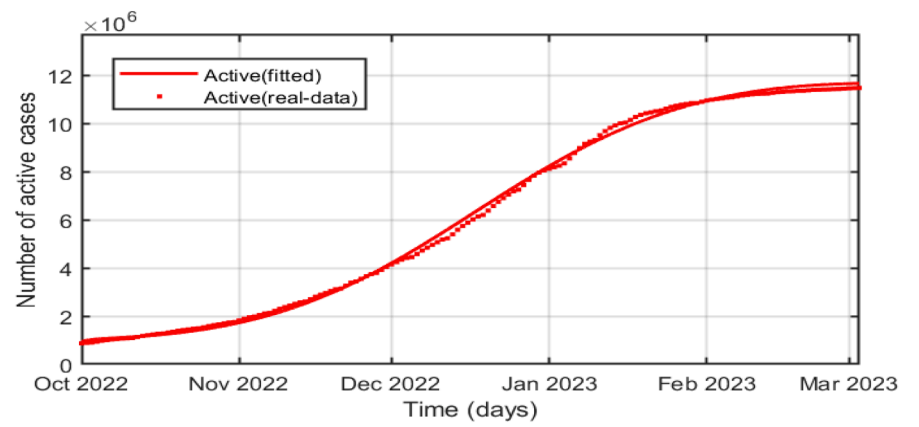
$\Xi(0) = 123951696, W(0) = 25856655, V(0) = 74371017, P(0) = 2156232, Z_e(0) = 86249, Z_c(0) = 862, Z_{oc}(0) = 6899, Z_o(0) = 86249, L(0) = 86249, M(0) = 86250, N(0) = 20393893, O(0) = 40404, Q(0) = 4489$. The simulations are conducted for the period (October 01, 2022, to March 03, 2023) (Worldometers.info (2023)). To simplify the model, the hospitalization and infection rates are considered the same for the omicron variant, variants other than omicron, and co-infection. The obtained results from the proposed model and its comparison with real data are shown in Figs. 2A, 2B, 2C. The simulation results follow the real-life trend for COVID-19 cases. Fig. 3 represents the dynamics of proposed model for sub-populations such as susceptible, exposed, infected with variants other than omicron, infected with omicron variant, infected with comorbidities, deaths with comorbidities, hospitalized, quarantined, deaths without comorbidities, diabetes, and vaccinated population.

4.1. Impact of comorbidities

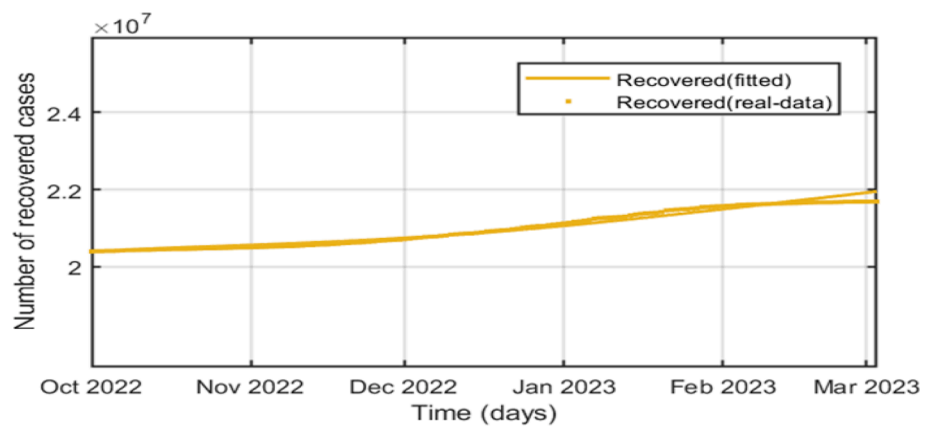
Figs. 4A, 4B, & 4C represent hospitalized cases, people with co-infection infection, and the number of death cases (with comorbidities), respectively. The results demonstrate that the number of hospitalized patients, infectious and comorbidities, and deaths with co-infections increase with an increase in the proportion of infectious diseases with comorbidities. The results are consistent with the study of Muniyappa and Gubbi (2020) and Rawshani et al. (2021), which divulge that people with diabetes have more tendency to hospitalization and death as compared to non-diabetic individuals. Fig. 5 demonstrates the impact of the co-infection rate on various sub-populations. The simulated results show that the number of infections with or without comorbidities increases with the increase in co-infection rate. The

Table 2
Best-fitted parameters for model (2.1).

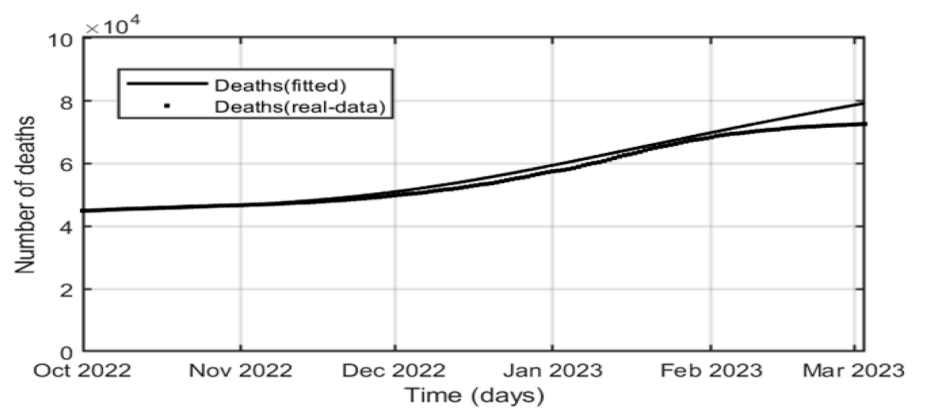
Parameter	Value	Parameter	Value
ϵ_1	0.001	ϖ, ζ_2, ζ_1	0.6711
ϑ	0.4383	σ	0.0031
ϑ_1	0.3506	ϑ_3	0.0035
n_j	0.006265	π_j	0.58
h	0.1123	ϑ_2	0.0044
ϵ	0.000409	m	0.02
ϵ_2	0.0001	δ_1	0.0015
u	0.0166	u_1	0.015
η	$6.4746e^{-5}$	χ	$3.237e^{-5}$
m_1	0.003	$\Phi_2(\tau)$	[0.0000, 0.0000, 0.0185]
δ	0.003	$\Phi_1(\tau)$	[0.0000, 0.0013, 4.6307]
$\Psi(\tau), \Psi_1(\tau)$	[0.0015, 0.0173, 61.0777]	$\Phi(\tau)$	[0.0001, 0.0026, 9.2614]
Θ	4012.6868		



(A)

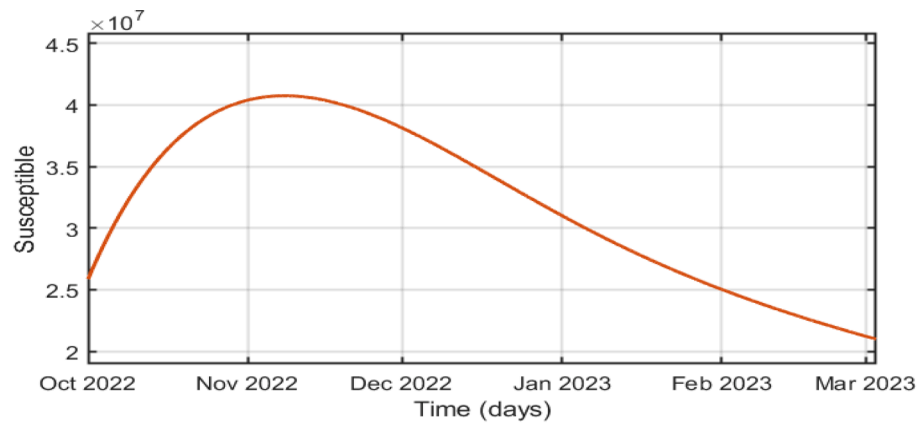


(B)

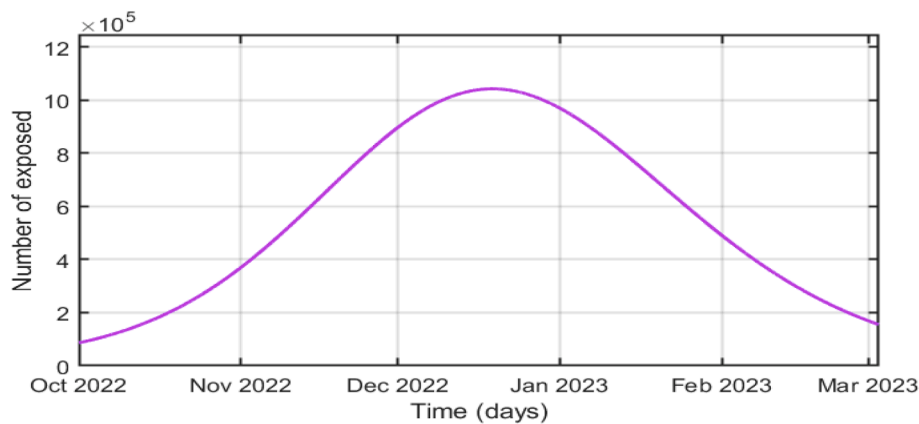


(C)

Fig. 2. Comparison between real reported and simulated cases. A) Infectious, B) Recovered, and C) Deaths.



(A)



(B)

Fig. 3. Dynamics of sub-population A) Susceptible, B) Exposed, C) Infected with variants other than omicron, D) Infected with omicron variant, E) Infected with comorbidities, F) Deaths with comorbidities, G) Hospitalized, H) Quarantined, I) Deaths without comorbidities, J) Diabetes, and K) Vaccination.

number of hospitalized and death cases also increases with an increase in co-infection rate, which made healthcare facilities and resources extensive. Excess burden on healthcare has led to higher mortality for COVID-19 cases.

4.2. Impact of hospitalization rate

The results in Fig. 6 demonstrate that the number of infectious decreases with an increase in hospitalization rate, which helps in declining the total number of death cases. It also delays the risk of transmission. When infectious with comorbidities, total infectious with symptoms, active cases, and hospitalized cases are simulated for different values of hospitalization rates as shown in Fig. 6. It has been noticed that community transmission has increased significantly with a decline in hospitalization rates. On the other hand, improving the hospitalization rate (by providing better healthcare guidance to people with comorbidities and increasing the testing for COVID-19) curtails community transmission. Moreover, Fig. 6 also investigates the death cases with comorbidities and total deaths for various values of hospitalization rate. The simulated results show a decline in death cases with an increase in the hospitalization rate, which suggests that improving the hospitalization rate helps reduce the number of death cases. The results in Fig. 6C

advocate that at the same hospitalization rate of $\zeta_1 = 0.1007$, the number of infectious with comorbidities would constrain the present resources due to the high proportion of the increase in infectious with comorbidities.

4.3. Impact of quarantine rate

Fig. 7 represents the impact of quarantine or self-isolation. It exhibits that the total number of infections increases with a decrease in quarantine rate and decreases with an increase in quarantine rate. The total number of deaths also decreases with an increase in quarantine or self-isolation rates, which helps in declining the mortality. Figs. 7E, 7F, 7G, & 7H show infectious with comorbidities, infectious (omicron variant), infectious (variant other than omicron), and total number of infectious, respectively for distinct values of quarantine rates. It is noticed that transmission increases significantly with a decrease in quarantine rates. On the contrary, increasing the quarantine or self-isolation rate (by creating awareness for infectious people with and without comorbidities about social distancing, self-isolation, quarantine, etc.) curtails community transmission. Furthermore, Fig. 7 also investigates the death cases with comorbidities, without comorbidities, total deaths, and hospitalized cases for distinct values of quarantine rates. The simulated

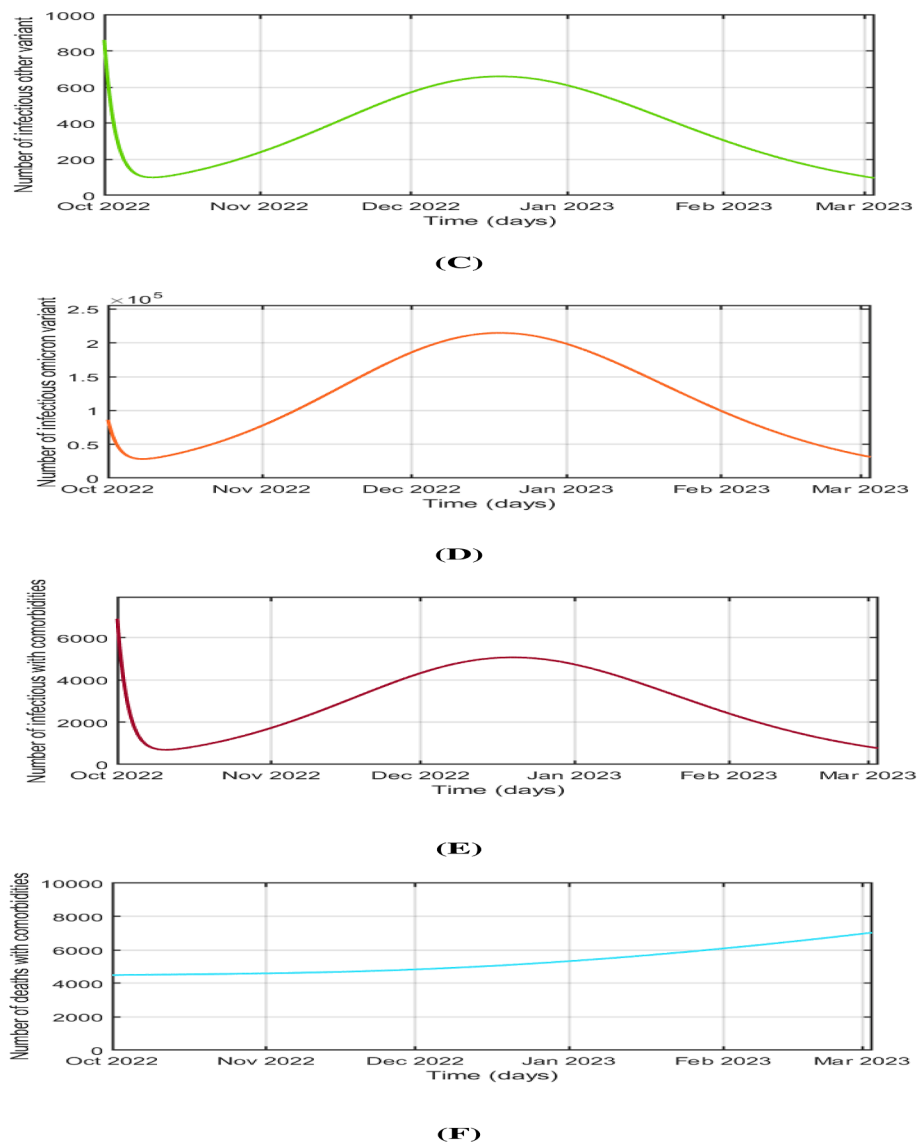


Fig. 3. (continued).

results show a decline in death cases with an increase in the quarantine rate, which suggests that revamping the quarantine or self-isolation helps in decreasing the number of death cases. The results in Figs. 7E, 7F, 7G, & 7H advocate that by reducing the quarantine rate, the number of infectious increases predominantly, and it constrains the present resources due to the high proportion of the rise in contagious.

4.4. Impact of vaccination

Vaccination is a crucial way to control the pandemic. Thus, the efficacy of vaccination corresponding to various doses, including booster dose, needs to be evaluated for a better understanding of disease dynamics. To study the various vaccination doses, we considered different vaccination rates and efficacy for doses as listed in Table 3. Fig. 8 shows the impact of different vaccination doses on novel coronavirus disease. The efficacy of the first dose of vaccine is 58% with a vaccination rate of 0.006265. The vaccination rate decreases slightly for the second dose to

0.006227 while vaccine efficacy improves to 68%. The vaccine efficacy increases to around 92% for booster dose with a decrease in the vaccination rate to 0.0053.

Figs. 8A, 8B, 8C, & 8D represent the exposed, infectious with other variant, infectious with omicron variant, and infectious with comorbidities, respectively. The simulation corresponds to the cases when there is no vaccine, first dose of vaccine, second dose of vaccine, and the case of booster dose is implemented.

Fig. 8D shows that the diabetic population gets immunity from the vaccination. Fig. 8E represents the hospitalized cases for COVID-19. Moreover, Fig. 8F depicts the quarantined or self-isolated cases with vaccination. Figs. 8G, 8H, & 8J represent deaths with comorbidities, without comorbidities, and cumulative deaths for case of no vaccination and decrease with different vaccination doses. Fig. 8I shows that the total infection is controlled by vaccination.

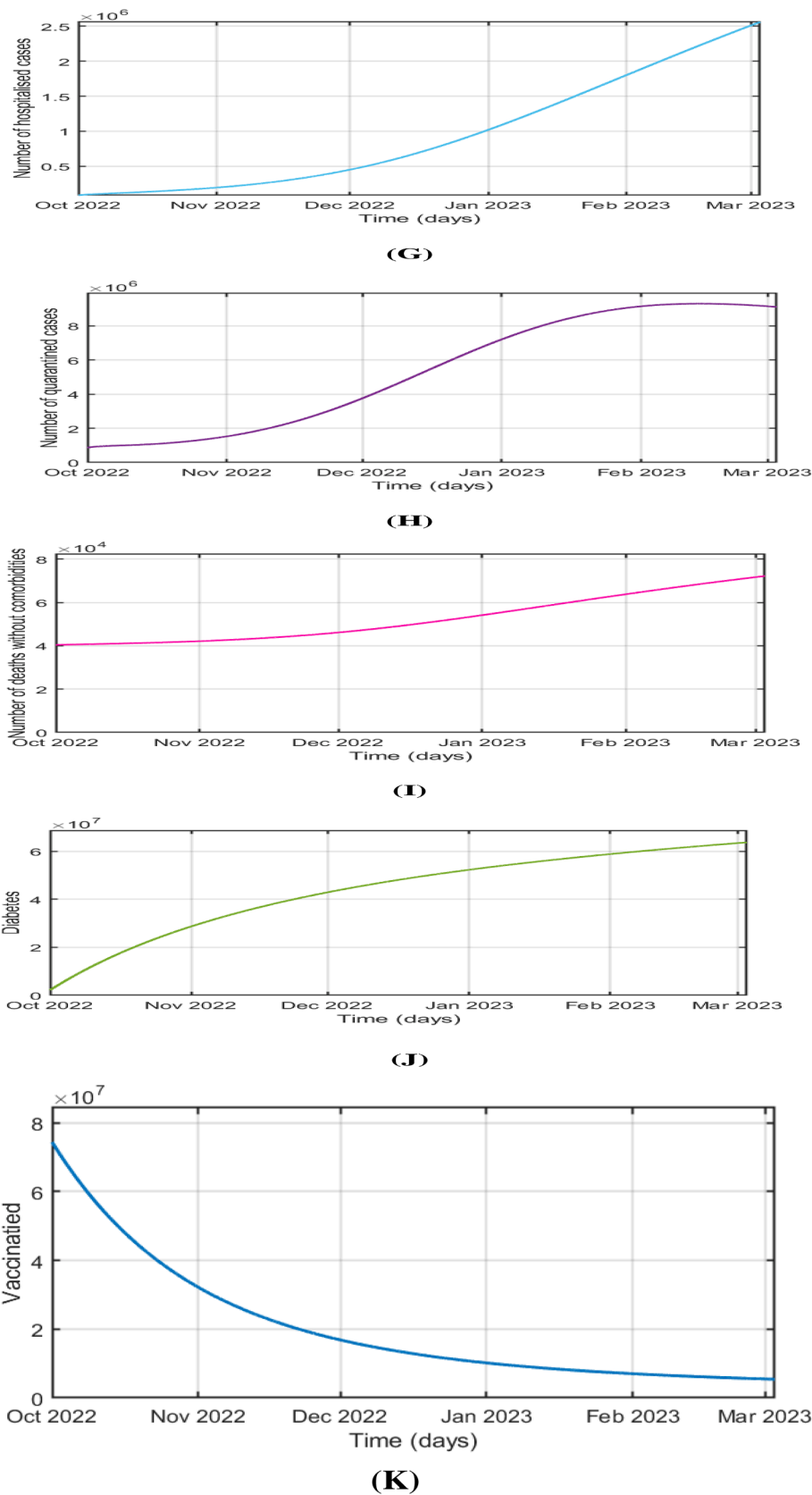
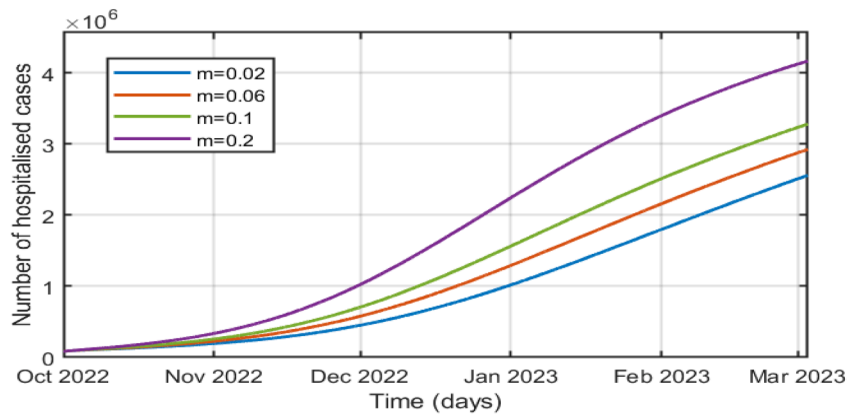
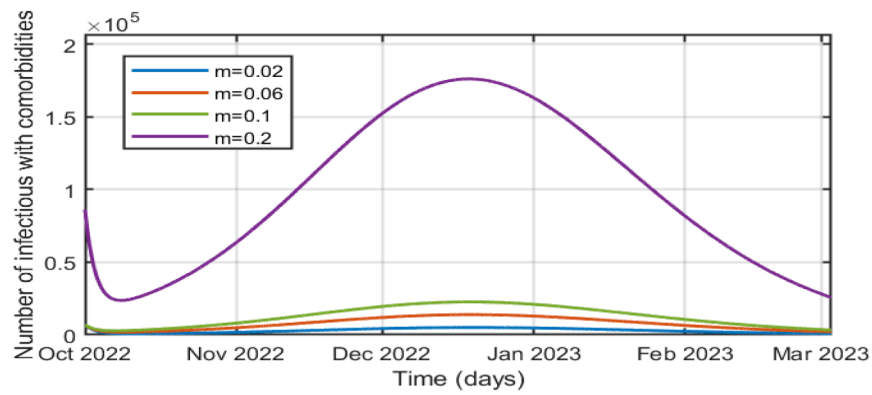


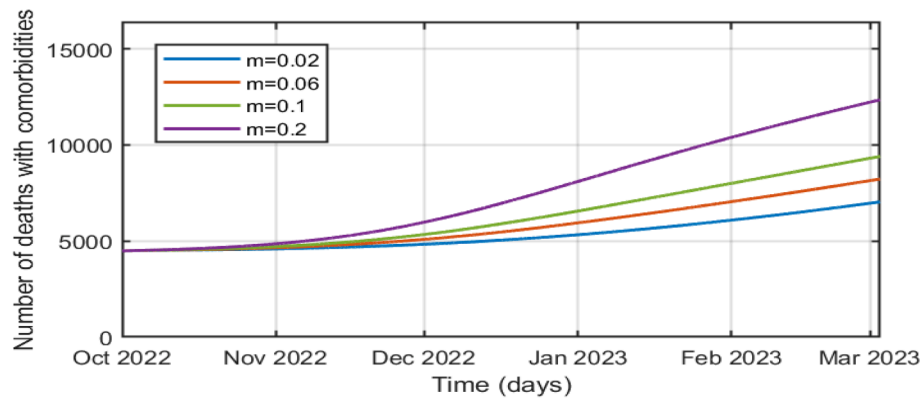
Fig. 3. (continued).



(A)

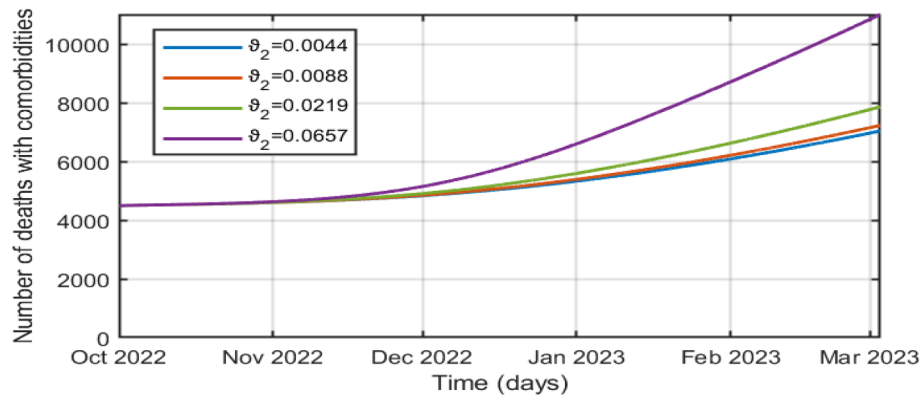


(B)

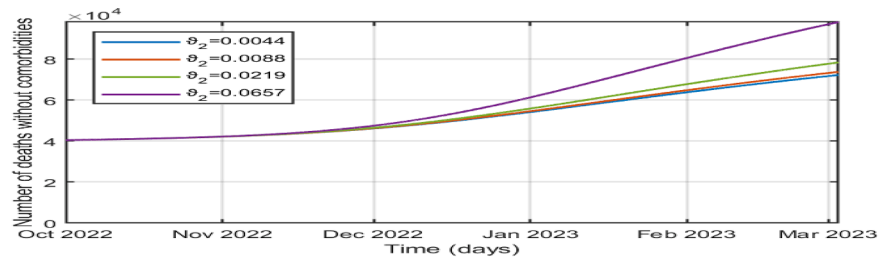


(C)

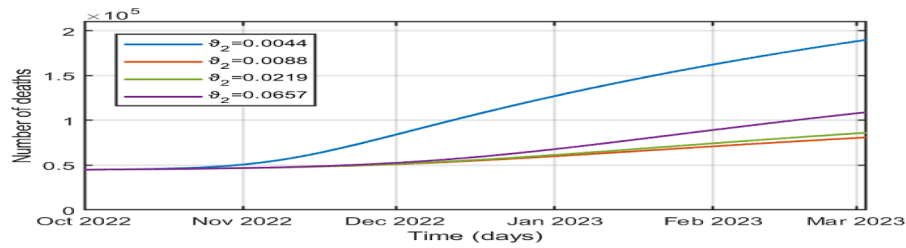
Fig. 4. Variation of probability for co-infection (m) A) Hospitalized, B) Infected with Comorbidities, and C) Deaths with Comorbidities.



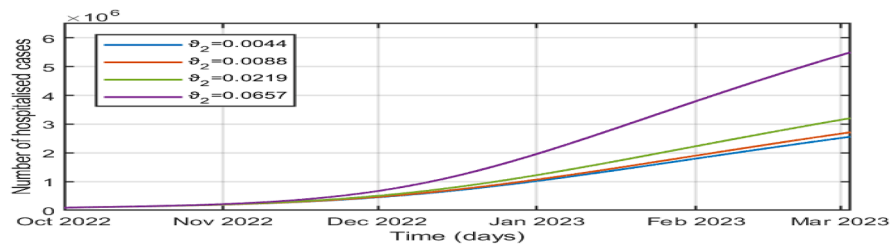
(A)



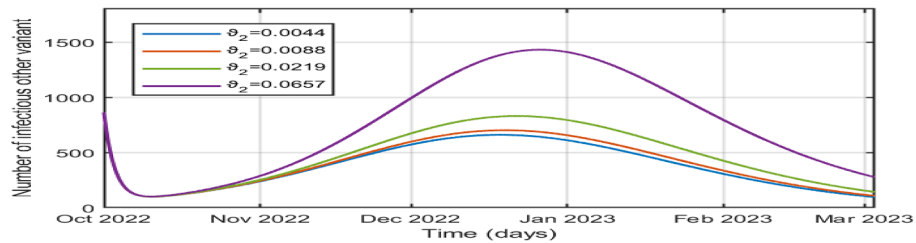
(B)



(C)

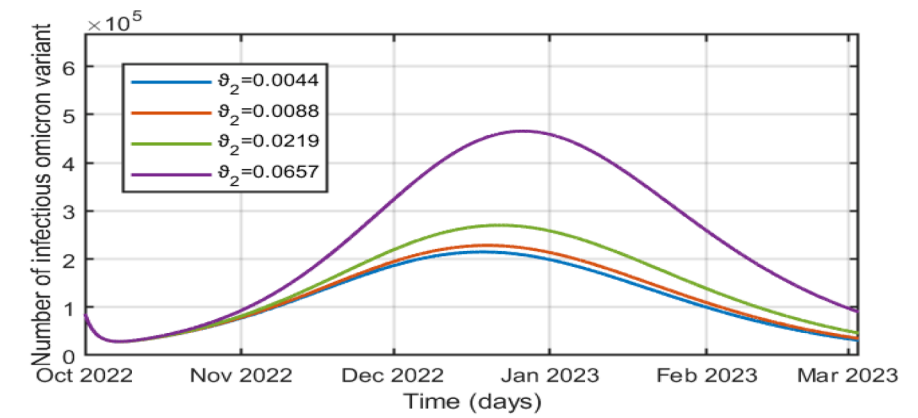


(D)

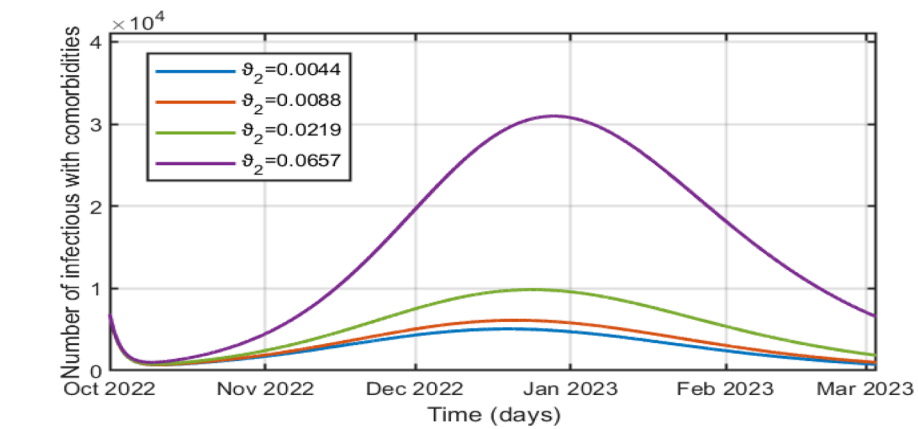


(E)

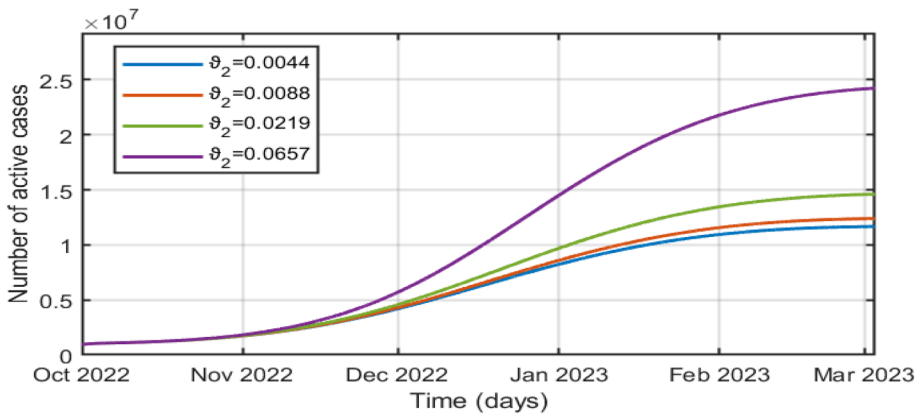
Fig. 5. Variation of infection rate for co-infection (ϑ_2) A) Deaths with comorbidities, B) Deaths without comorbidities, C) Deaths, D) Hospitalized, E) Infected with variants other than omicron, F) Infected with omicron variant, G) Infected with comorbidities, and H) Active cases.



(F)

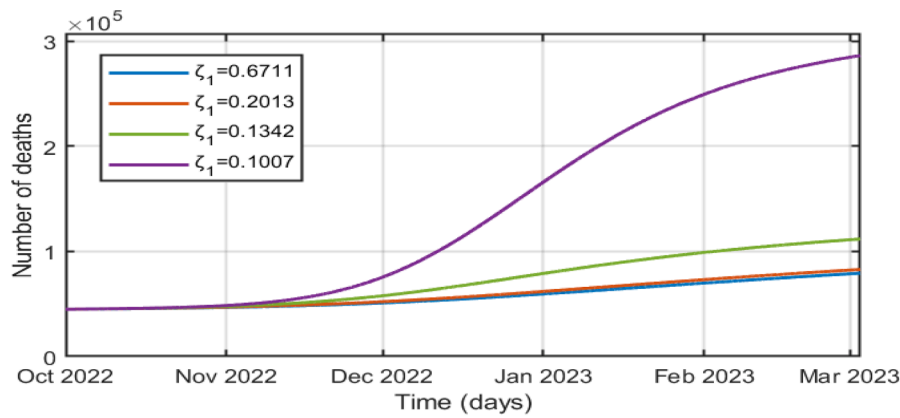


(G)

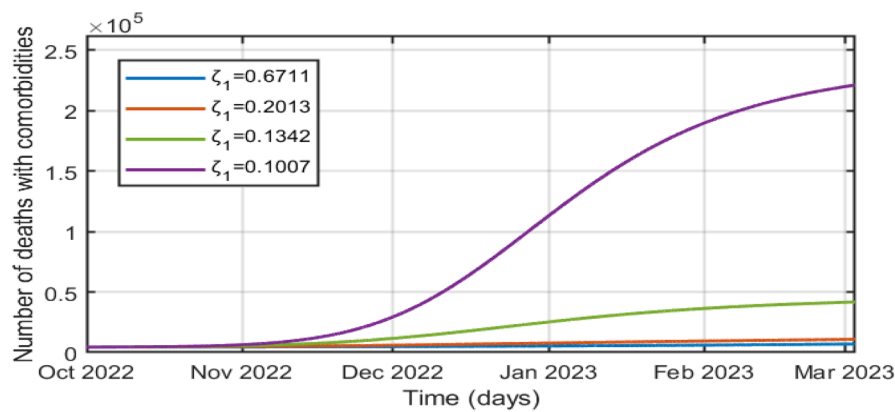


(H)

Fig. 5. (continued).



(A)



(B)

Fig. 6. Variation of hospitalization rate (ζ_1) for different sub-populations A) Deaths, B) Deaths with Comorbidities, C) Infected with Comorbidities, D) Total infectious (symptomatic) E) Active cases, and F) Hospitalized.

5. Discussion and conclusion

This work developed a new model with omicron variants and variants other than omicron as different compartments. Furthermore, the impact of vaccination doses is evaluated in detail. The ground analysis of the model (2.1) is done, which is utilized to gain insight into the effect of hospitalization, quarantine, or self-isolation on novel coronavirus disease in a group of people, some of them with co-infection. To formulate the model, we divided the whole population into susceptible, exposed, infectious omicron variants, infectious variants other than omicron, infectious with comorbidities, quarantined, hospitalized, diabetes, recovered, vaccinated, and deaths with and without comorbidities compartments. The force of infection combines infectious with comorbidities, with the omicron variant and variants other than omicron. The spread of novel COVID-19 is assessed for individuals with and without symptoms. The obtained solutions of the model at DFE points are locally and globally asymptotically stable whenever $R_0 < 1$. This entails that the disease may be eradicated regardless of its initial infection in the community, with some individuals having comorbidities. The basic reproduction number depends on the infection rate for symptomatic and

asymptomatic individuals, vaccine efficacy, vaccination rate, latent period, hospitalization rate, and those following standard operating procedures (SOPs). The findings indicate that if individuals without comorbidities fail to adhere to the SOPs, the healthcare system has been overburdened due to the increase in novel coronavirus cases, particularly among individuals with chronic conditions. The findings depict the dynamics of the proposed model. It is observed that the recovered, infected (active cases), and deaths fit well when compared with actual data. Figs. 8A, 8B, 8C, & 8D demonstrate the exposed, infectious with other variant, infectious with omicron variant, and infectious with comorbidities, correspondingly. In cases when there is no vaccine, the first dose of vaccine, a second dose of vaccine, and the booster dose are deliberated. It is illustrated from the results that the infectious population rose quickly in case of no vaccination, decreased for the first and second doses of vaccination, and the curve gets even flattened in the case of booster dose. Fig. 8D represents that the diabetic people also receive immunity from the vaccination. It is observed from Fig. 8D that the diabetic population infected with COVID-19 declines with the first dose, a second dose, and even curtailed with booster dose. Fig. 8E shows the hospitalized cases for COVID-19. The result exhibits that the

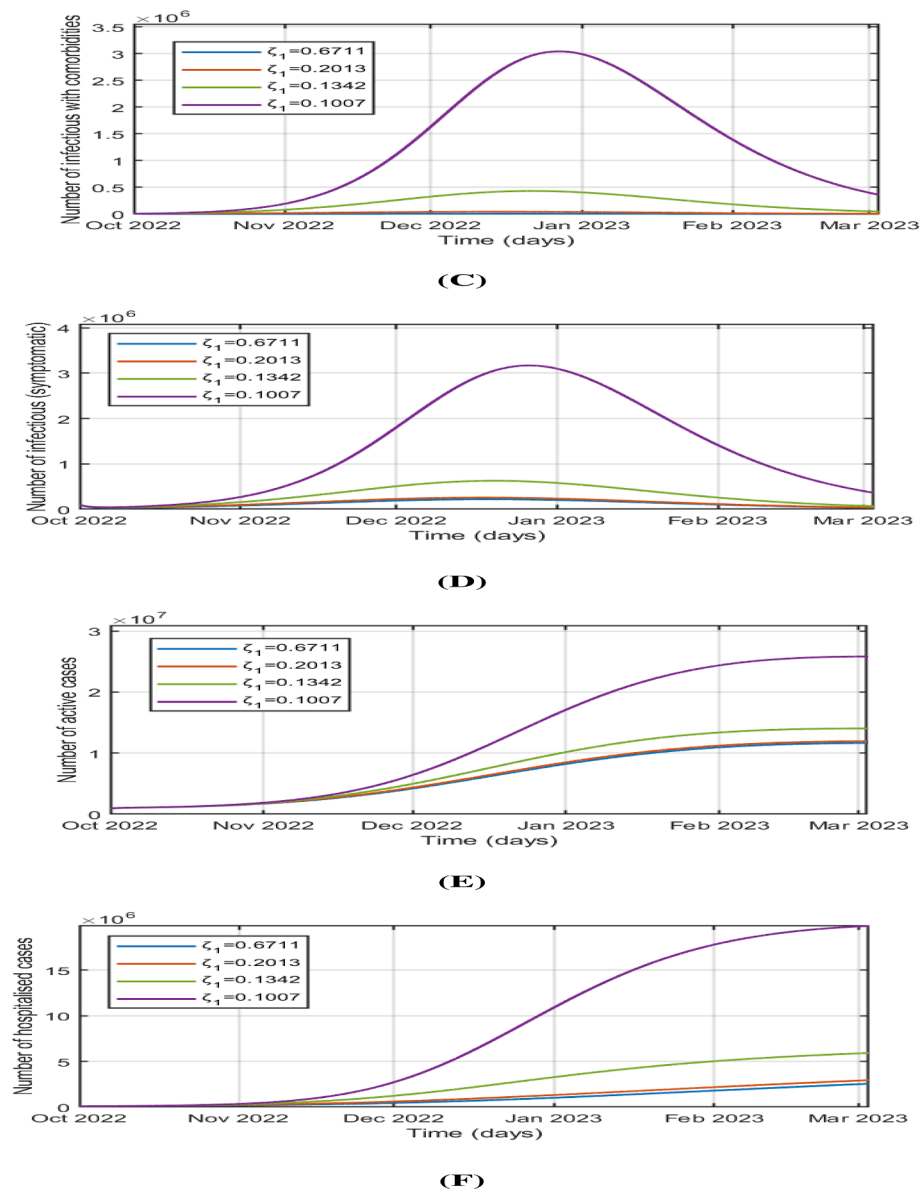


Fig. 6. (continued).

hospitalized patients are excessive in case of no vaccination. Hospitalized patients less after the first vaccination dose and better with complete vaccination. It is abated by taking the booster dose, which helps boost the individual's immunity. A decrease in hospitalized cases lowers the burden on the healthcare system. The number of patients with the omicron variant declines rapidly with the execution of different vaccine doses see Fig. 8C. Asymptomatic patients are also diminished by vaccination (Fig. 8A). Furthermore, the quarantined or self-isolated individuals are also reduced through vaccination Fig. 8F, reducing wellness concerns. The deaths with comorbidities, without comorbidities, and total deaths increased in case of no vaccination and declined with implementation of vaccination (Figs. 8G, 8H, & 8J). Fig. 8I represents that the cumulative infection is regulated through vaccination. Therefore, it is accomplished that vaccination has lowered contaminant

transmission and helped lower the severity and fatality. Furthermore, the simulation results also reveal that the number of hospitalized, infected individuals with comorbidities and deaths with comorbidities increases with an increase in the probability of co-infection (m). In several countries, comorbidities have resulted in a significant increase in the prevalence and number of deaths (Kyrychko et al. (2020)). Simulation results demonstrate the significance of early detection, reporting, and effective treatment in preventing novel coronaviruses. It is also noticed that for the hospitalization rate ($\zeta_1 = 0.1007$), the number of infected with comorbidities increases in a higher proportion. The increased prevalence of infectious diseases with comorbidities has limited the available resources. In the scenario where people who have comorbidities report for treatment and hospitalization effectively, the epidemic curve spiked lower. The findings in Fig. 5 suggest that the

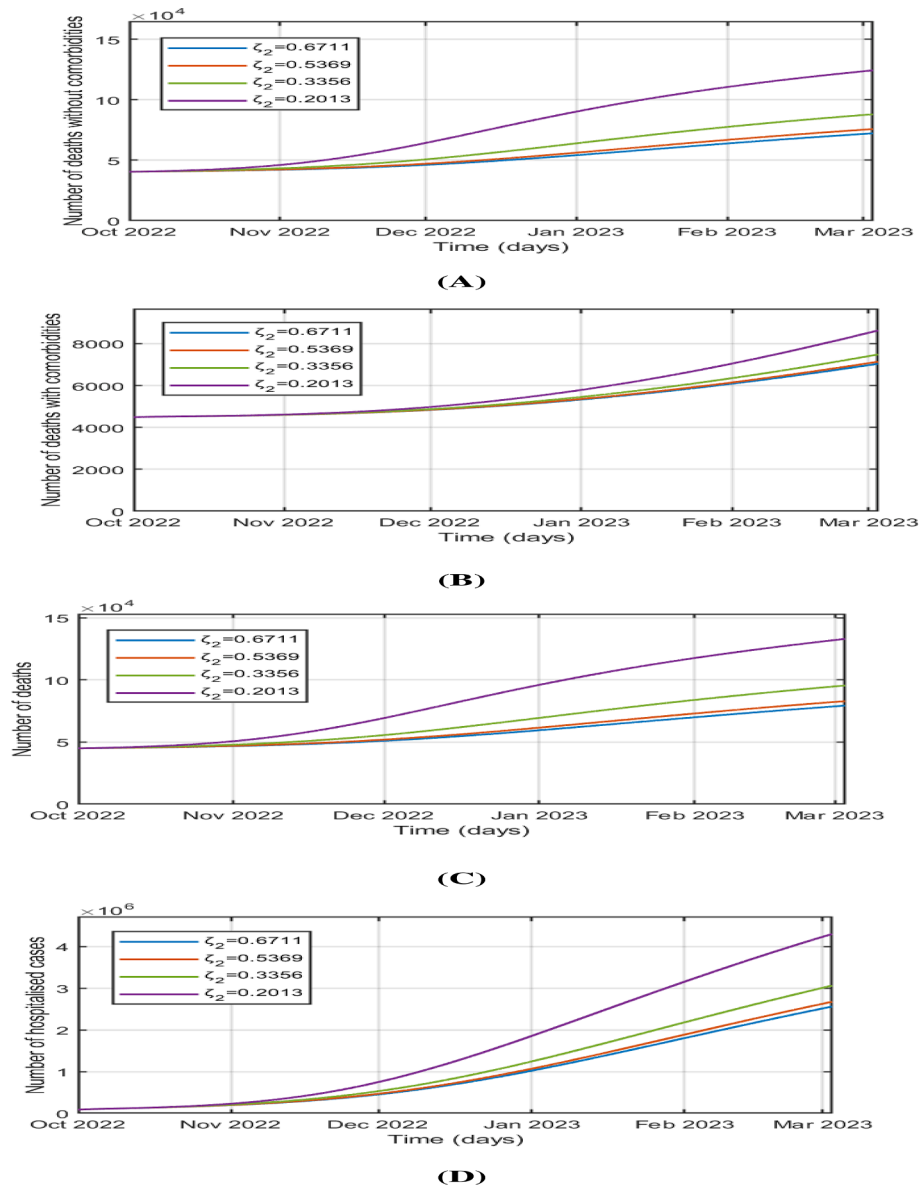


Fig. 7. Variation of quarantine or self-isolation rate (ζ_2) A) Deaths without comorbidities, B) Deaths with comorbidities, C) Deaths, D) Hospitalized, E) Infected with variants other than omicron, F) Infected with omicron variant, G) Infected with comorbidities, and H) Active cases.

number of infections is lowered by lowering the co-infection rate. The results reveal the effectiveness of SOP (quarantine, self-isolation, etc.). The importance of vaccination policy and its doses are also realized from the findings. The study on the effectiveness of vaccinations demonstrated the critical role that vaccination played in stopping the spread of the virus. Significant reductions in infectious and symptomatic infectious outbreaks resulted from higher vaccination efficacy rates. The decline in hospital admissions demonstrated the importance of increased vaccination efficacy in lessening the burden on healthcare institutions. Additionally, the study showed how the booster dose affected hospitalized, symptomatic, and asymptomatic infectious patients with diabetes by highlighting the sharp decline in infections. Effective vaccination is helpful for people with diabetes as well. The number of people with diabetes lowers with the first, second, and booster dose. Moreover, vaccination is capable of decreasing the

infection with the omicron variant.

In conclusion, this work presented a new model for diabetes and coronavirus with an omicron variant and vaccination with booster dose. We have evaluated the efficiency of the proposed model both analytically and numerically. The proposed study is validated by employing real data from Japan. Furthermore, we recognized the impact of parameters through sensitivity analysis. Several parameters (ζ_1, ζ_2, m , and ϑ_2) are examined for different population classes. The findings concluded that the diabetic population infected with COVID-19 (symptomatic) increases with the increase of parameters (m and ϑ_2) and decreases with a higher hospitalization rate. The impact of vaccination is demonstrated for the first, second, and booster doses. The findings demonstrate that when dose-1 of the vaccine is received, the asymptomatic infectious decreases compared to the case of no vaccination. The infection (asymptomatic) decreases more with the second dose and

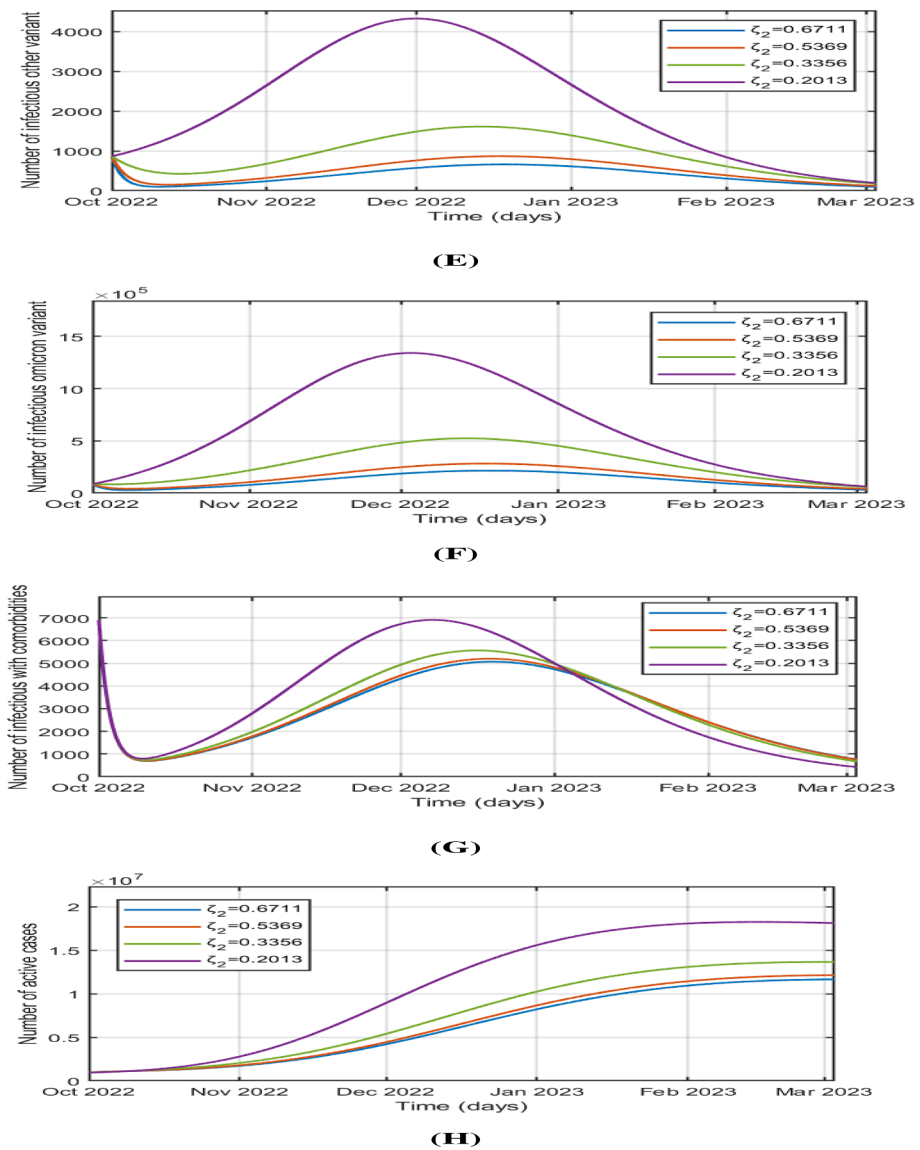


Fig. 7. (continued).

Table 3
Vaccination rates and efficacy model (2.1).

Vaccination rates (π_i)	Value	Vaccination efficacy (η_i)	Value
π_0	0	η_0	0
π_1	0.006265	η_1	0.58
π_2	0.006227	η_2	0.68
π_3	0.0053	η_3	0.92

booster dose. The results show that the number of people who have not had either dose-1 or dose-2 of vaccination and are infected with the omicron variant increases rapidly compared to when either dose-1, dose-2, or booster dose is received. It is concluded that the diabetic infectious (co-infected) decreases significantly when receiving the booster dose. Therefore, it is concluded that vaccination is of utmost importance to control the group's spread. The findings also demonstrate the

significance of sticking to SOPs, early detection, and hospitalization of coronavirus patients, particularly those without chronic disease, to reduce community transmission. Vaccination of eligible individuals at large is recommended for future waves to control the spread of disease. Furthermore, this model can be utilized for another disease of similar nature. The effects of delay, fractional derivatives, economic aspects, etc., can also be studied by extending the model.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

Data will be made available on request.

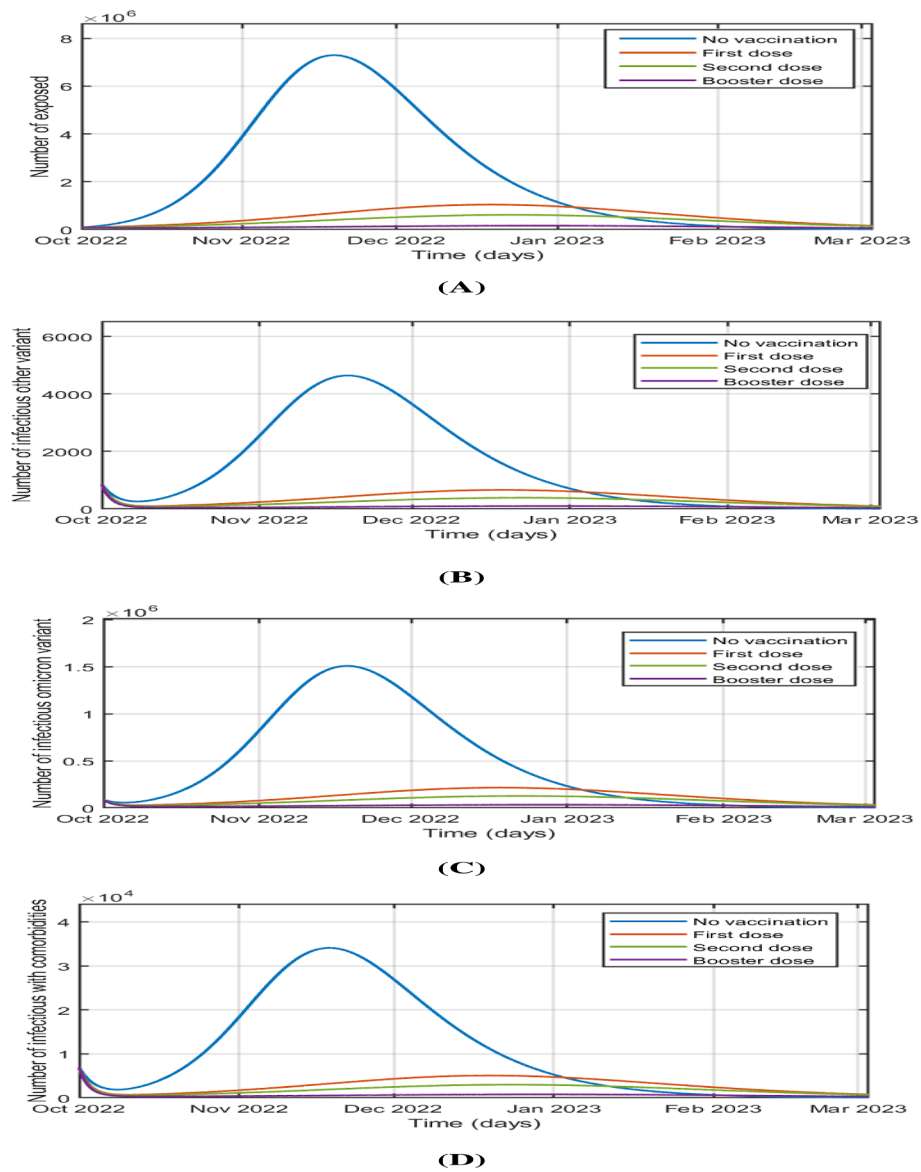


Fig. 8. Dynamics of sub-population for vaccination doses (A) Exposed, (B) Infected with variants other than omicron, (C) Infected with omicron variant, (D) Infected with comorbidities, (E) Hospitalized, (F) Quarantined, (G) Deaths without comorbidities, (H) Deaths with comorbidities, (I) Active cases, and (J) Deaths.

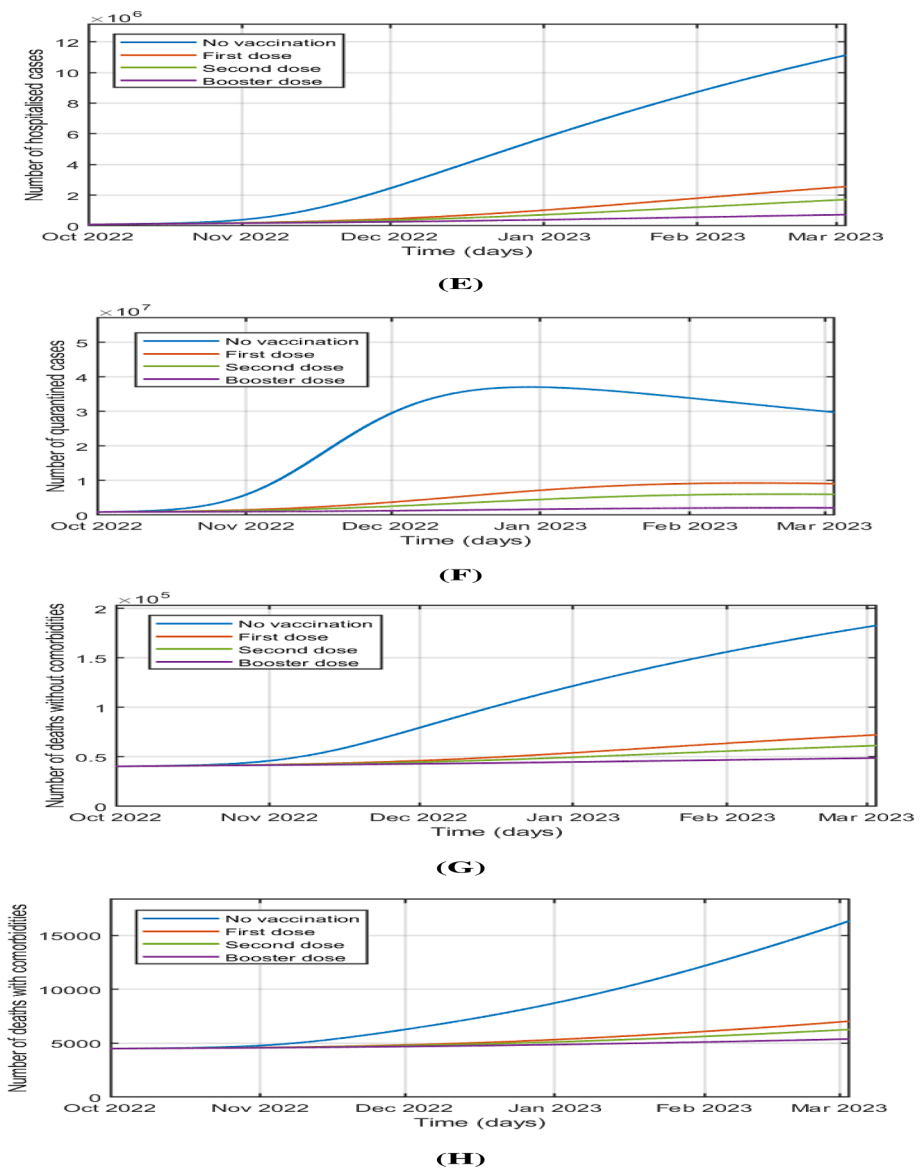
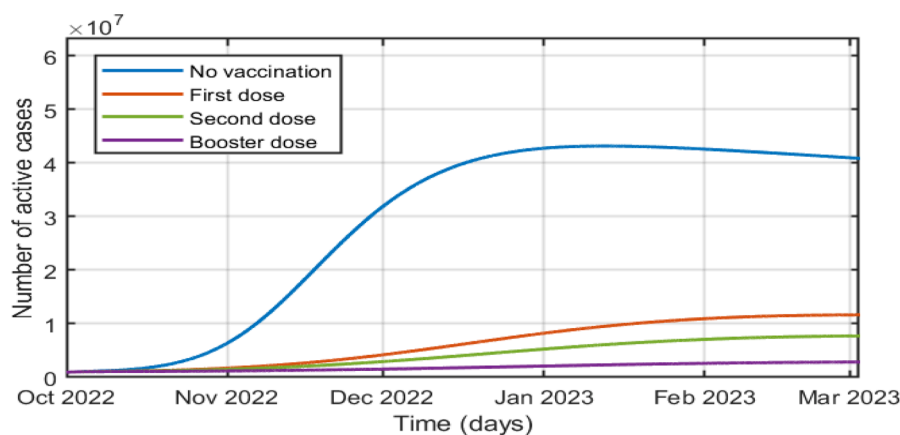
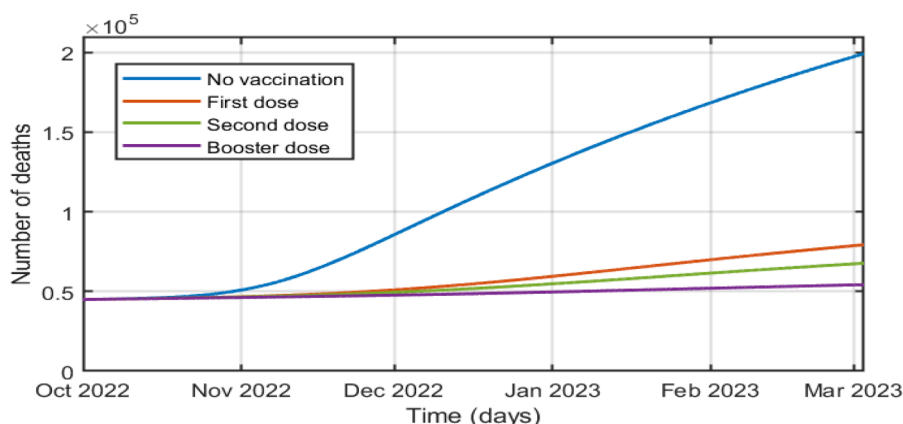


Fig. 8. (continued).



(I)



(J)

Fig. 8. (continued).

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References

- Abdullah, F., Myers, J., Basu, D., Tintinger, G., Ueckermann, V., Mathebula, M., Jassat, W., 2022. Decreased severity of disease during the first global omicron variant covid-19 outbreak in a large hospital in tshwane, South Africa. *Int. J. Infect. Diseases* 116, 38–42.
- Agrawal, U., Bedston, S., McCowan, C., Oke, J., Patterson, L., Robertson, C., Sheikh, A., 2022. Severe covid-19 outcomes after full vaccination of primary schedule and initial boosters: pooled analysis of national prospective cohort studies of 30 million individuals in england, northern ireland, scotland, and wales. *The Lancet* 400 (10360), 1305–1320.
- Anand, M., Danumjaya, P., Rao, P.R.S., 2023. A nonlinear mathematical model on the covid-19 transmission pattern among diabetic and non-diabetic population. *Math. Comput. Simul.* 210, 346–369.
- S. Anusha, S. Athithan, Mathematical modelling co-existence of diabetes and covid-19: Deterministic and stochastic approach (2021).
- Auvigne, V., Vaux, S., Le Strat, Y., Schaeffer, J., Fournier, L., Tamandjou, C., du Châtelet, I.P., 2022. Severe hospital events following symptomatic infection with sars-cov-2 omicron and delta variants in france, december 2021–january 2022: a retrospective, population-based, matched cohort study. *E Clinical Medicine* 48, 101455.
- Badawi, A., Ryoo, S.G., 2016. Prevalence of diabetes in the 2009 influenza a (h1n1) and the middle east respiratory syndrome coronavirus: a systematic review and meta-analysis. *Journal of Public Health Research* 5 (3), 733–739.
- Barron, E., Bakhal, C., Kar, P., Weaver, A., Bradley, D., Ismail, H., Valabhji, J., 2020. Associations of type 1 and type 2 diabetes with covid-19-related mortality in england: a whole-population study. *The Lancet Diabetes & Endocrinology* 8 (10), 813–822.
- Bekiros, S., Kouloumpou, D., 2020. Sbdien: a new mathematical model of infectious disease dynamics. *Chaos, Solitons & Fractals* 136, 109828.
- Bhadoria, A.S., Devi, S., Gupta, N., 2022. Modelling and analysis of a seiqr model on covid-19 pandemic with delay. *Modeling Earth Systems and Environment* 8 (3), 3201–3214.
- Bichara, D., Iggidr, A., Sallet, G., 2014. Global analysis of multi-strains sis, sir and msir epidemic models. *Journal of Applied Mathematics and Computation* 44 (1–2), 273–292.
- Carey, I.M., Critchley, J.A., DeWilde, S., Harris, T., Hosking, F.J., Cook, D.G., 2018. Risk of infection in type 1 and type 2 diabetes compared with the general population: a matched cohort study. *Diabetes Care* 41 (3), 513–521.
- Castillo-Chavez, C., Feng, Z., Huang, W., 2002. On the computation of r_0 and its role in global stability. *IMA Volumes in Mathematics and Its Applications* 125, 229–250.
- CDC NDSS, National diabetes statistics report, available from: <https://www.cdc.gov/diabetes/pdfs/data/statistics/nationaldiabetes-statistics-report.pdf>, Accessed 3 August 2022 (2020).
- Centers for Disease Control and Prevention, Coronavirus disease 2019 (covid-19), available on <https://www.cdc.gov/coronavirus/2019-ncov/index.html> (2020).
- Cheyne, E., 2020. Generalized seir epidemic model (fitting and computation). <https://doi.org/10.5281/ZENODO.3911854>.
- Chitnis, N., Hyman, J., Cushing, J., 2008. Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model. *Bull. Math. Biol.* 70 (5), 1272–1296.
- Czeisler, M.E., Barrett, C.E., Siegel, K.R., Weaver, M.D., Czeisler, C.A., Rajaratnam, S.M., Bullard, K.M., 2021. Health care access and use among adults with diabetes during the covid-19 pandemic—united states, february–march 2021. *Morb. Mortal. Wkly Rep.* 70 (46), 1597–1602.
- Das, P., Upadhyay, R.K., Misra, A.K., Rihan, F.A., Ghosh, D., 2021. Mathematical model of covid-19 with comorbidity and controlling using non-pharmaceutical interventions and vaccination. *Nonlinear Dyn.* 106 (2), 1213–1227.

- Davies, A., Kassanjee, R., Rousseau, P., Morden, E., Johnson, L., Solomon, W., Hsiao, Y., Hussey, H., Meintjes, G., Paleker, M., Jacobs, T., Raubenheimer, P., Heekes, A., Dane, P., Bam, L., Smith, M., Preiser, W., Pienaar, D., Mendelson, M., Boule, A., 2022. Outcomes of laboratory-confirmed sars-cov-2 infection in the omicron-driven fourth wave compared with previous waves in the western cape province, south africa. *Tropical Medicine & International Health* 27 (6), 564–573. <https://doi.org/10.1111/tmi.13752>.
- Diekmann, O., Heesterbeek, J.A.P., Roberts, M.G., 2010. The construction of next-generation matrices for compartmental epidemic models. *Journal of the Royal Society Interface* 47, 7873–7885.
- El-Sayed, A.M.A., El-Mesiry, A.E.M., El-Saka, H.A.A., 2007. On the fractional-order logistic equation. *Applied Mathematics Letters* 20, 817–823.
- Esper, F.P., Adhikari, T.M., Tu, Z.J., Cheng, Y.W., El-Haddad, K., Farkas, D.H., Rubin, B. P., 2023. Alpha to omicron: disease severity and clinical outcomes of major sars-cov-2 variants. *J. Infect. Dis.* 227 (3), 344–352.
- GISAID, Tracking of hcov 19 variants, available online: <https://www.re3data.org/repository/r3d100010126>, accessed on 11 December 2022 (2022).
- Gram, M.A., Emborg, H.D., Schelde, A.B., Friis, N.U., Nielsen, K.F., Moustsen-Helms, I.R., Hansen, C.H., 2022. Vaccine effectiveness against sars-cov-2 infection or covid-19 hospitalization with the alpha, delta, or omicron sars-cov-2 variant: A nationwide danish cohort study. *PLoS Medicine* 19 (9), e1003992.
- Holman, N., Knighton, P., Kar, P., O'Keefe, J., Curley, M., Weaver, A., Valabhji, J., 2020. Risk factors for covid-19-related mortality in people with type 1 and type 2 diabetes in england: a population-based cohort study. *The Lancet Diabetes & Endocrinology* 8 (10), 823–833.
- Jørgensen, S.B., Nygård, K., Kacelnik, O., Telle, K., 2022. Secondary attack rates for omicron and delta variants of sars-cov-2 in norwegian households. *J. Am. Med. Assoc.* 327 (16), 1610–1611.
- Kavya, K., Veerasha, P., Baskonus, H.M., Alsulami, M., 2024. Mathematical modeling to investigate the influence of vaccination and booster doses on the spread of omicron. *Commun. Nonlinear Sci. Numer. Simul.* 130, 107755.
- Khan, M.S., Ozair, M., Hussain, T., Gómez-Aguilar, J.F., 2021. Bifurcation analysis of a discrete-time compartmental model for hypertensive or diabetic patients exposed to covid-19. *The European Physical Journal Plus* 136 (8), 1–26.
- Kouidere, A., Youssoufi, L.E., Ferjouchia, H., Balatif, O., Rachik, M., 2021. Optimal control of mathematical modeling of the spread of the covid-19 pandemic with highlighting the negative impact of quarantine on diabetics people with cost-effectiveness. *Chaos, Solitons & Fractals* 145, 110777.
- Kumari, P., Singh, S., Singh, H.P., 2022. Dynamical analysis of covid-19 model incorporating environmental factors. *Iranian Journal of Science and Technology, Transaction A: Science* 46 (6), 1651–1666.
- Kumari, P., Singh, H.P., Singh, S., 2023. Global stability of novel coronavirus model using fractional derivative. *Computational and Applied Mathematics* 42, 346.
- Kyrychko, Y.N., Blyuss, K.B., Brovchenko, I., 2020. Mathematical modeling of the dynamics and containment of covid-19 in ukraine. *Scientific Reports* 10 (1), 19662.
- Landstra, C.P., de Koning, E.J.P., 2021. Covid-19 and diabetes: Understanding the interrelationship and risks for a severe course. *Frontiers in Endocrinology* 12, 649525.
- Li, H., Tian, S., Chen, T., Cui, Z., Shi, N., Zhong, X., Zheng, J., 2020. Newly diagnosed diabetes is associated with a higher risk of mortality than known diabetes in hospitalized patients with covid-19. *Diabetes, Obesity and Metabolism* 22 (10), 1897–1906.
- M.M. Lima-Martínez, C. Carrera Boada, M.D. Madera-Silva, W. Marín, M. Contreras, Covid-19 and diabetes: A bidirectional relationship. covid-19 y diabetes mellitus: una relación bidireccional, *Clinica e Investigación en Arteriosclerosis: Publicación Oficial de la Sociedad Española de Arteriosclerosis* 33 (3) (2021) 151–157.
- Liu, J., Bai, R., Chai, Z., Cooper, M.E., Zimet, P.Z., Zhang, L., 2022. Low-and middle-income countries demonstrate rapid growth of type 2 diabetes: An analysis based on global burden of disease 1990–2019 data. *Diabetologia* 65 (8), 1339–1352.
- Liu, S., Jiang, C., Liu, Y., Zhang, Y., Qiu, X., Luo, J., Wang, J., 2023. The effectiveness of covid-19 vaccination against all-cause mortality in patients with type 2 diabetes mellitus: The observation during the initial period of the cancellation of the “dynamic zero policy” in mainland china. *Diabetes Res. Clin. Pract.* 200, 110694.
- Massard, M., Eftimie, R., Perasso, A., Sausseureau, B., 2022. A multi-strain epidemic model for covid-19 with infected and asymptomatic cases: Application to french data. *J. Theor. Biol.* 545, 111117.
- MathWorks LSQCURVEFIT, Function details for lsqcurvefit - atlas user documentation, accessed 20 October 2023 (2016). <https://www.atlas.aei.uni-hannover.de/valentin.frey/profile/file73.html>.
- McConeghy, K.W., White, E.M., Blackman, C., Santostefano, C.M., Lee, Y., Rudolph, J.L., Gravenstein, S., 2022. Effectiveness of a second covid-19 vaccine booster dose against infection, hospitalization, or death among nursing home residents-19 states, march 29–july 25, 2022. *Morb. Mortal. Wkly Rep.* 71 (39), 1235–1238.
- McGurnaghan, S.J., Weir, A., Bishop, J., Kennedy, S., Blackburn, L.A., McAllister, D.A., McCoubrey, J., 2021. Risks of and risk factors for covid-19 disease in people with diabetes: a cohort study of the total population of scotland. *The Lancet Diabetes & Endocrinology* 9 (2), 82–93.
- Muniyappa, R., Gubbi, S., 2020. Covid-19 pandemic, coronaviruses, and diabetes mellitus. *American Journal of Physiology-Endocrinology and Metabolism* 318 (5), E736–E741.
- Negi, S., Rana, P., Sharma, N., Khatri, M., 2022. A novel seiahr compartment model for accessing the impact of vaccination, intervention policies, and quarantine on the covid-19 pandemic: a case study of most affected countries brazil, india, italy, and usa. *Computational and Applied Mathematics* 41 (7), 305.
- Niemi, M.E.K., Daly, M.J., Ganna, A., 2022. The human genetic epidemiology of covid-19. *Nat. Rev. Genet.* 23 (9), 533–546.
- N.I. of Infectious Diseases, About sars cov 2 variants, available online: <https://www.niid.go.jp/niid/ja/2019-ncov/2551-cepr/10745-cepr-topics.html>, accessed on 11 December 2022 (2022).
- Nogrady, B., 2020. What the data say about asymptomatic covid infections. *Nature* 587 (7835), 534–535.
- T. Nyberg, N.M. Ferguson, S.G. Nash, H.H. Webster, S. Flaxman, N. Andrews, S. Thelwall, Comparative analysis of the risks of hospitalization and death associated with sars-cov-2 omicron (b. 1.1. 529) and delta (b. 1.617. 2) variants in england: a cohort study, *The Lancet* 399 (10332) (2022) 1303–1312.
- Ogata, T., Tanaka, H., Tanaka, E., Osaki, N., Noguchi, E., Osaki, Y., Wada, K., 2022. Increased secondary attack rates among the household contacts of patients with the omicron variant of the coronavirus disease 2019 in japan. *International Journal of Environmental Research and Public Health* 19 (13), 8068.
- Okoye, J.O., 2020. Attitudinal, regional and sex related vulnerabilities to covid-19: Considerations for early flattening of curve in nigeria. *Medical Journal of the Islamic Republic of Iran* 34, 61.
- Omame, A., Sene, N., Nometa, I., Nwakanma, C.I., Nwafor, E.U., Iheonu, N.O., Okuonghae, D., 2021. Analysis of covid-19 and comorbidity co-infection model with optimal control. *Optimal Control Applications & Methods* 42 (6), 1568–1590.
- Omame, A., Nwajeri, U.K., Abbas, M., Onyenegecha, C.P., 2022. A fractional order control model for diabetes and covid-19 co-dynamics with mittag-leffler function. *Alexandria Engineering Journal* 61 (10), 7619–7635.
- Özköse, F., Yavuz, M., 2022. Investigation of interactions between covid-19 and diabetes with hereditary traits using real data: A case study in turkey. *Comput. Biol. Med.* 141, 105044.
- Patel, S.Y., McCoy, R.G., Barnett, M.L., Shah, N.D., Mehrotra, A., 2021. Diabetes care and glycemic control during the covid-19 pandemic in the united states. *JAMA Internal Medicine* 181 (10), 1412–1414.
- Rana, P., Sharma, N., 2022. The modeling and analysis of the covid-19 pandemic with vaccination and treatment control: a case study of maharashtra, delhi, uttarakhand, sikkim, and russia in the light of pharmaceutical and non-pharmaceutical approaches. *European Physical Journal Special Topics* 231 (18–20), 3629–3648.
- Rao, P.R.S., Kumar, M.N., 2015. A dynamic model for infectious diseases: the role of vaccination and treatment. *Chaos, Solitons & Fractals* 75, 34–49.
- Rawshani, A., Kjölhede, E.A., Rawshani, A., Sattar, N., Eeg-Olofsson, K., Adiels, M., Rosengren, A., 2021. Severe covid-19 in people with type 1 and type 2 diabetes in sweden: A nationwide retrospective cohort study. *The Lancet Regional Health-Europe* 4.
- Sheikh, A., Kerr, S., Woolhouse, M., McMenamin, J., Robertson, C., Simpson, C.R., McCowan, C., 2022. Severity of omicron variant of concern and effectiveness of vaccine boosters against symptomatic disease in scotland (leave ii): a national cohort study with nested test-negative design. *Lancet. Infect. Dis* 22 (7), 959–966.
- Shi, Q., Zhang, X., Jiang, F., Zhang, X., Hu, N., Bimu, C., Wang, W., 2020. Clinical characteristics and risk factors for mortality of covid-19 patients with diabetes in wuhan, china: A two-center, retrospective study. *Diabetes Care* 43 (7), 1382–1391.
- Singh, A.K., Gillies, C.L., Singh, R., Singh, A., Chudasama, Y., Coles, B., Khunti, K., 2020. Prevalence of co-morbidities and their association with mortality in patients with covid-19: a systematic review and meta-analysis. *Diabetes, Obesity and Metabolism* 22 (10), 1915–1924.
- Smith, D.J., Hakim, A.J., Leung, G.M., Xu, W., Schluter, W.W., Novak, R.T., Hersh, B.S., 2022. Covid-19 mortality and vaccine coverage—hong kong special administrative region, china, january 6, 2022–march 21, 2022. *Morb. Mortal. Wkly Rep.* 71 (15), 545–548.
- Ssebuliba, J., Nakakawa, J.N., Ssematimba, A., Mugisha, J., 2022. Mathematical modelling of covid-19 transmission dynamics in a partially comorbid community. *Partial Differential Equations in Applied Mathematics* 5, 100212.
- Takahashi, Y., Tanaka, H., Koga, Y., Takiguchi, S., Ogimoto, S., Inaba, S., Ogata, T., 2023. Change over time in the risk of death among japanese covid-19 cases caused by the omicron variant depending on prevalence of sublineages. *International Journal of Environmental Research and Public Health* 20 (4), 2779.
- Theparod, T., Kreabkhonhth, P., Teparos, W., 2023. Booster dose vaccination and dynamics of covid-19 pandemic in the fifth wave: An efficient and simple mathematical model for disease progression. *Vaccines* 11 (3), 589.
- Tyagi, S., Martha, S.C., Abbas, S., Debouche, A., 2021. Mathematical modeling and analysis for controlling the spread of infectious diseases. *Chaos, Solitons & Fractals* 144, 110707.
- Wang, C., Liu, B., Zhang, S., Huang, N., Zhao, T., Lu, Q.B., Cui, F., 2023. Differences in incidence and fatality of covid-19 by sars-cov-2 omicron variant versus delta variant in relation to vaccine coverage: A world-wide review. *J. Med. Virol.* 95 (1), e28118.
- World Health Organization, Classification of omicron (b.1.1.529): Sars cov 2 variant of concern, available online: [https://www.who.int/news/item/26-11-2021-classification-of-omicron-\(b.1.1.529\)-sars-cov-2-variant-of-concern](https://www.who.int/news/item/26-11-2021-classification-of-omicron-(b.1.1.529)-sars-cov-2-variant-of-concern), accessed on 11 December 2022 (2022).
- World Health Organization, WHO covid-19 dashboard: Cases, accessed: 2024-07-25 (2024). URL <https://data.who.int/dashboards/covid19/cases>.
- Worldometers.info, accessed on 03 March 2023 (2023). <https://www.worldometers.info/coronavirus/>.
- Xue, L., Jing, S., Zhang, K., Milne, R., Wang, H., 2022. Infectivity versus fatality of sars-cov-2 mutations and influenza. *International Journal of Infectious Diseases* 121, 195–202.
- Yao, X.I., Han, L., Sun, Y., He, D., Zhao, S., Ran, J., 2023. Temporal variation of excess deaths from diabetes during the covid-19 pandemic in the united states. *J. Infect. Public Health* 16 (4), 483–489.
- Zhou, F., Yu, T., Du, R., Fan, G., Liu, Y., Liu, Z., Cao, B., 2020. Clinical course and risk factors for mortality of adult inpatients with covid-19 in wuhan, china: a retrospective cohort study. *The Lancet* 395 (10229), 1054–1062.