



A Model of COVID-19 Epidemic Based on Vaccination and Treatment

Fahdah Alshammari¹, Alaa Mustafa^{1,*}, Ehssan Omer², Fatima Alnoor³

¹ Department of Biology, Faculty of Science and Arts-RAFHA, Northrn Border University, Arar 73213 Kingdom of Saudi Arabia

² Department of Mathematics, Faculty of Science -Arar, Northrn Border University, Arar 75211 Kingdom of Saudi Arabia

³ Department of Mathematics, Faculty of Science and Arts-TURIAF, Northrn Border University, Arar 75211 Kingdom of Saudi Arabia

Abstract. This article presents a mathematical model of the COVID-19 transmission mechanism, considering therapeutic interventions like immunization and recovery or treatment. The model shows that the disease-free and endemic equilibriums are globally asymptotically stable when effective reproduction numbers are less than or larger than unity. The critical vaccination threshold depends on the vaccine's ability to prevent or cure the illness. The model predicts the effectiveness of vaccination based on factors like vaccination efficiency, scheduling, and relaxation of social measures. The subsiding of the epidemic as vaccination is implemented depends on the scale of relaxation of social measures.

2020 Mathematics Subject Classifications: 37N25

Key Words and Phrases: Covid-19 Epidemic, Mathematical Modeling, Vaccination, Social measures, Transmission

1. Introduction

Years of study and testing are frequently needed to create vaccinations that are safe and effective against infectious agents. In comparison, COVID-19 vaccine development and distribution took less than a year [18]. The effectiveness of the epidemiological models suggested for the COVID-19 outbreak may be impacted by the answers to the many unanswered issues created by this rapid development. For instance, vaccine efficiency rates vary depending on the level and length of immunization for various groups [23], [24], [13], [17]. According to the epidemic status of the vaccinated individuals—vulnerable,

*Corresponding author.

DOI: <https://doi.org/10.29020/nybg.ejpam.v16i4.4805>

Email addresses: bfahdah1402@hotmail.com (F. Alshammari),
alal200949@yahoo.com (A. Mustafa), ehssanomerphdd@gmail.com (E. Omer),
fattoshaa1988@gmail.com (F. Alnoor)

infected, or having received vaccine doses—we will examine the efficacy of vaccination [22]. Since the start of the COVID-19 pandemic [[11], [14], [12], [5], [21], [4][19]], mathematical modeling has been utilized to examine the reliability of parameters, forecast its progression, and contrast various.

Despite these limitations, a first simplified approach can lead to a model, making it possible to predict effective vaccination coverage at the population level that will prevent the appearance of successive epidemic waves. [15]

Daily vaccination data, even if they are global and unrefined (for example, [14] by age group or social classification), make it possible to better understand the effect of vaccination policy and test the consequences of changes in this policy to improve its effectiveness[19]. We use an epidemic model to understand the complex interactions between epidemic control and epidemic data. [[18], [9]] Our model considers the changes in public health policy [[1], [20], [12]], such as confinement, social distancing measures, etc., through the time-dependent transmission rate in the model. The data consists of the daily number of reported cases and the daily number of second doses of vaccine. We refer to [[13], [17], [9], [5] [15]] for more results on the subject.

In the study, we propose a new model for vaccination implementation. We can connect the model with vaccination to a model without vaccination.

We will find a simple transformation for the epidemic data to combine the daily reported case data and the cumulative number of vaccinated individuals[12]. We can use the model to explore controlling the dynamics of virus propagation, for example, by rapidly slowing down an epidemic wave.

In this new model, we will explicitly take into account the variable corresponding to the size of the vaccinated population, and we will simulate the increasing efficacy of several vaccination scenarios. Then, we will apply our model to the COVID-19 epidemic. By applying the maximum principle, optimal control of the model can mitigate the spread of COVID-19[7].

2. Mathematical COVID-19 Epidemic models.

$$\begin{aligned}
 S'(t) &= -\rho(t)[I(t) + \omega N(t)]S(t) - \varepsilon Va_{Data}(t) \frac{S(t)}{U_v(t)} \\
 E'(t) &= -\rho(t)[I(t) + \omega N(t)]S(t) - \beta E(t) - \varepsilon Va_{Data}(t) \frac{e(t)}{U_v(t)} \\
 I'(t) &= \beta E(t) - \sigma I(t) - \varepsilon Va_{Data}(t) \frac{E(t)}{U_v(t)} \\
 N'(t) &= \sigma(1 - r)I(t) - \gamma N(t) - \varepsilon Va_{Data}(t) \frac{E(t)}{U_v(t)} \\
 R'(t) &= \sigma r I(t) - \gamma R(t)
 \end{aligned} \tag{1}$$

where, when t , $S(t)$ is the proportion of susceptible, individual health, $E(t)$ is the proportion of those who were exposed, $I(t)$ is the proportion of infected yet asymptomatic

persons, $N(t)$ is the proportion of symptomatic infected people who go undetected, and $R(t)$ is the proportion of symptomatic infected people who have been reported[10]. $U_v(t)$ is the proportion of unvaccinated people. The biological interpretations of all parameters are provided in Table 1.

The primary information is added to the system (1).

$$S(t_1) = S_1, E(t_1) = E_1, I(t_1) = I_1, N(t_1) = N_1, R(t_1) = R_1 \tag{2}$$

The model features, $\varnothing(t)$ is the transmission rate that varies with time, $1/\beta$ is the median amount of the exposure time, $1/\sigma$ is the maximum range of the infectious period without symptoms, And for the sake of simplicity, we divide the group of symptomatic infectious persons into two fractions: the fraction $0 \leq r \leq 1$ showing severe symptoms and the fraction $1 - r$ showing moderate symptoms, which is considered to be undiagnosed. For both unreported and reported symptomatic individuals, the amount $1/\gamma$ represents the average length of the symptomatic infectious period. [23] The factor $(0 \leq \omega \leq 1)$ represents the relative contributions of undetected and unreported clinical infectious persons to the infection of susceptible individuals. The potency of the vaccine is the parameter $0 \leq \varepsilon \leq 1$. This implies that the vaccination is totally effective at $\varepsilon = 1$ and completely ineffective at $\varepsilon = 0$, respectively. The equation is satisfied by the total number of removed persons $t \rightarrow M(t)$, immunized (recovered or vaccinated), and/or dead[6].

$$M'(t) = \gamma[R(t) + N(t)] + eV_a(t) \left[\frac{S(t) + E(t) + I(t) + N(t)}{U_v(t)} \right] \tag{3}$$

In this instance, $V_a(t)$ there is steady stream of properly vaccinated people. It implies that

$$\int_{t_1}^t V_A(\alpha).d\alpha$$

Is the overall vaccination rate between time points a and b[2]. Since no people received vaccinations at the beginning of the disease (i.e., for $t = t_1$), we can assume that the total population P at time t_1 is

$$P = S_1 + E_1 + I_1 + R_1 + N_1$$

The total number of people who have received vaccinations is provided by

$$LV_a(t_1) = 0 \quad \text{and} \quad LV'_a(t) = V_a(t),$$

it is comparable to

$$LV_a(t) = \int_{t_1}^t V_A(\alpha).d\alpha$$

The number of people that aren't vaccinations is

$$U_v(t) = P - LV_a(t)$$

It's expected that a fraction $0 < r \leq 1$ of infectious persons is reported at the end of the asymptomatic infectious period. Therefore, the following connection links the epidemic model to the total number of reported cases $LR(t)$, for $t \geq t_1$, and $LR_0(t_1) = LR_0$.

$$LR'_a(t) = \sigma r I(t), \tag{4}$$

Beginning at time t_1 , we define the fraction of people who are not fully immunized at time t as

$$G(t) = \exp\left(\varepsilon \int_{t_1}^t \frac{P - V_a(\alpha)}{P - V_a(\alpha)} d\alpha\right).$$

Then,

$$G(t) = \exp(\varepsilon(\ln [P - V_a(t)] - \ln [P - V_a(t_1)]))$$

Therefore,

$$G(t) = \left(\frac{P - LV_a(t)}{P - LV_a(t_1)}\right)^\varepsilon \tag{5}$$

If $LV_a(t)$ is the total number of recipients of the second dose vaccination. When $G(0) = 1$, the function $t, W(t)$ is non-increasing. Define

$$LR_a(t) = \int_{t_1}^t \frac{LR'_a(\alpha)}{G(\alpha)} d\alpha \tag{6}$$

2.1. Measuring the data transmission.

The parameters $\omega, \beta, \sigma, \gamma, r, S_1, E_1, I_1, N_1$, and the five subsequent equations $t \geq t_1$ fully indicate the data transmission.

$$\varnothing(t) = \frac{\hat{\varnothing}(t)}{G(t)} \tag{7}$$

therefore, at $t \geq t_1$

$$\hat{I}(t) = \frac{I(t)}{G(t)}, \hat{N}(t) = \frac{N(t)}{G(t)}, \text{ and } \hat{\varnothing}(t) = \varnothing(t)G(t).$$

$$\hat{\varnothing}(t) = \frac{1}{\hat{\varnothing}(t)\omega\hat{N}(t)} \times \frac{\hat{L}E(t) + \beta\hat{L}E'(t)}{E_1 + S_1 - \hat{L}E'(t) + \beta\hat{L}E'(t)}$$

and

$$\hat{\varnothing}(t) = \frac{LR'_a(t)}{\sigma r},$$

$$\widehat{L}E(t) = \frac{1}{\beta\sigma r}(\widehat{L}R'_a(t) - \sigma r I_1 + \sigma(LR'_a)'(t))$$

$$\hat{N}(t) = e^{-\gamma(t-t_1)}N_1 + \int_{t_1}^t e^{-\gamma(t-s_0)}\frac{1-r}{r}\widehat{L}R'_a(s_1).ds_1$$

The data used in Formula (6) to define $(LR_a)(t)$ are those represented by functions $t \rightarrow LR_a(t)$ the cumulative number of reported cases and $t \rightarrow V_a(t)$ resources spent on second doses of vaccination (t) [12].

We arrive at the following equation to define the rate of transmission as a function of the total number of reported cases $t \rightarrow LR_a(t)$ and the total number of immunized people $t \rightarrow LV_a(t)$ [11].

2.2. Statistics homogenized by $G(t)$

The everyday number of identified cases normalized by $G(t)$, or the percentage of those who weren't effectively immunized at time t ,

$$\widehat{LR}_a(t)' = \frac{LR_a(t)'}{G(t)} = LR_a(t)' \left(\frac{P}{P - LV_a(t)} \right)^\epsilon$$

Case frequency on a regular schedule standardized by $W(t)$ is

$$\widehat{LR}_a(t) = \int_{t_1}^t \frac{LR_a'(\alpha)}{G(\alpha)} d\alpha \left(\frac{P}{P - LV_a(t)} \right)^\epsilon$$

2.3. Reproduction Numbers

Utilizing the Formula, we can get the daily transmission rate $t \rightarrow \rho(t)$. [16] The issue of the immediate reproduction numbers may then be considered by utilizing model (6) (see [24] for more information). We apply our approach to calculate the transmission rate and take into account the immediate reproduction number with vaccination to explore the role of vaccination in the COVID-19 data [13].

$$R_V(t) = \frac{\rho(t)S(t)}{\gamma\sigma} \times \gamma + \omega\sigma(1 - r) \tag{8}$$

The reproduction rate with vaccination

$$R_V^1(t) = \frac{\rho(t)G(t)S_1}{\gamma\sigma} \times \gamma + \omega\sigma(1 - r) \tag{9}$$

The reproduction rate Without vaccination

$$R^1(t) = \frac{\rho(t)S_1}{\gamma\sigma} \times (\gamma + \omega\sigma(1 - r)), \tag{10}$$

3. Mathematical Analysis of the Epidemic Model

In this section, we explain the COVID-19 model's boundedness, positiveness, epidemic and endemic stability, and fundamental reproduction frequency [4].

Theorem 1. *If $S_1 \geq 0, E_1 \geq 0, I_1 \geq 0, N_1 \geq 0, R_1 \geq 0$, then the solutions of system (1) remain Positiveness for all $t > 0$.*

Proof. Proof. We obtain the following from system (1.1)'s first equation:

$$\frac{ds}{dt} \geq -\omega S \tag{11}$$

$$\int ds \geq -\omega S \cdot dt$$

$$S(t) \geq S_1 e^{-\omega t} \tag{12}$$

Therefore, for any $t > 0, S(t)$ stays positive. We also get $E(t) \geq 0, I(t) \geq 0, N(t) \geq 0$, and $R(t) \geq 0$, using the remaining equations of system (1). Theorem 1 is verified.

Theorem 2. *All solutions of the proposed model with Positiveness initial conditions are bounded and*

$$P(t) \leq \frac{\rho}{\omega} \text{ for all } t > 0.$$

Proof. The overall population's growth rate may be expressed as the sum of all the equations in the system (1).

$$\frac{dP}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dN}{dt} + \frac{dR}{dt} \tag{13}$$

Equation (3) gives the following formula:

$$\frac{dP}{dt} = \rho - \omega P \tag{14}$$

The solution is obtained by integrating two sides of Equation (14) as follows:

$$P(t) = \frac{\rho}{\omega} + (P_1 - \frac{\rho}{\omega})e^{-\omega t}. \tag{15}$$

Therefore, for $t \rightarrow \infty$, we have that

$$P(t) \leq \frac{\rho}{\omega} \tag{16}$$

Thus, Theorem 1 as well as Equation (16) conclude in

$$0 \leq P(t) \leq \frac{\rho}{\omega}$$

Therefore, $S(t), E(t), I(t), N(t)$, and $R(t)$ are bounded. Theorem 2 is so verified

3.1. Epidemic equilibrium and the model's essential reproduction number

When all derivatives are set to zero with $I = 0$, the epidemic equilibrium Y^0 a system (1) is attained, which results in:

$$Y^0 = \frac{\rho}{\omega + w'}, 0, 0, 0, \frac{w\rho}{\omega(\omega + w)} \quad (17)$$

The disease-free equilibrium Y^0 is locally asymptotically stable if $R^1 < 1$, and unstable if $R^1 > 1$ [1].

3.2. Transmission rate

The model calculates the time-dependent transmission rate before the last date of daily reported cases using daily reported cases data. A rolling weekly average is used to smooth the data[17].

3.3. Vaccination rate

The study aims to determine the effectiveness of vaccination in removing susceptible individuals from infection risk. The daily number of vaccinated individuals and cumulative vaccination rate are graphed from December 2020 to June 2021. The efficiency of vaccination is assumed to be 98%, in TABLE 2 and the removal of susceptible is the fraction of the number vaccinated, excluding previously infected individuals. Future daily vaccination rates are chosen for illustration due to their uncertain values[3].

4. Result and Discussion

The Covid-19 pandemic was predicted using the suggested modeling technique. The Centers for Disease Prevention and Control have data accessible [8]. The number of fatalities as well as those retrieved and verified are among them. Daily assimilation of the overall mortality toll, active cases, and recovered cases took place.

4.1. The Number of Instantaneous Reproduction

On the fundamental reproduction number, we see essentially little effect on the vaccine's effectiveness. This results from compensating effects between $R(t)$ and $S(t)$, which are assessed to change the fixed number of cumulative reported cases. We hardly notice any change. It indicates that the cumulative infection rate is so low relative to the size of the whole population that the $R(t)$ remains essentially unchanged from $R^1(t)$ [22]. This indicates that the total number of infected people is insufficient to significantly affect the rate of basic reproduction. To put it another way, the changes in the population of vulnerable individuals are not big enough to be seen immediately.

The R^1 is shown by the blue curve $R(t)$. Given that the vaccine is entirely effective,

the instantaneous reproduction number should be understood as the instantaneous reproduction number in the absence of immunization. [2] The same explanation applies to $\varepsilon = 0.75, 0.5, 0.25$, and 0. This indicates that vaccination significantly affects the dynamics of the epidemic. The effectiveness of the vaccination is a key factor in this influence. We can see that without vaccination. The final peak of the red curve, which represents $R^1(t)$ about Theorem 2, is present for $\varepsilon = 0.75$ [9].

4.2. Impact of Vaccination

Began a three-phase COVID-19 immunization campaign on July 17, 2022, to December 20, 2022, making it the first Arab nation to introduce the Pfizer-BioNTech vaccine. [23] The first phase focuses on those over 65 and those who are most at risk for the disease. People over 50 and those with particular chronic conditions, such as diabetes and asthma, will be included in the second phase. In the third step, the vaccine will be administered to the remaining population. The Ministry has, however, identified people who shouldn't receive the vaccination. Women who are pregnant or nursing, those who want to get pregnant within the next two months, those who experience severe allergic reactions, and those who have been exposed to the virus within the last 90 days are among them[19].

4.3. Future Work and Modeling Expansions.

. Despite these flaws, the model effectively illustrates how vaccination policies affect epidemic dynamics, in part because precise formulas allow for the computation of crucial variables like transmission rate. In addition, the model permits the incorporation of other components, including age, immunization loss, and cross-immunization, when supported by actual data. The efficiency of the immunization strategy is weighed down by this most recent phenomenon, which merits more study. The vaccination policy may be changed in light of the response via cross-immunity against epitopes shared by several Corona viruses. Vaccination provides new immunity in addition to any potential pre-existing cross-immunity [19]. For instance, when age groups are taken into account, young people are those whose cross-immunity is still active.

5. Conclusions

The study presents an enhanced SEIR model for including vaccines in epidemic models, considering seven infection phases and vaccination. The model demonstrates non-negativity, boundedness, epidemic equilibrium, existence, distinctiveness of endemic equilibrium, and fundamental reproduction number. It incorporates COVID-19 dynamics, transmission, reported and unreported cases, and social distancing measures. The model predicts vaccination effectiveness in the US, with varying vaccination rates due to hesitancy and opposition. Future research will explore other countries and locations.

Table 1: Descriptions of parameters in system(1).

Parameter	Description	Value
ω	Unreported symptomatic infectious cases that can spread the disease	[25]
$\frac{1}{\beta}$	Average time expended having exposed	Estimated
$\frac{1}{\varphi}$	The average duration of the infectious period without indications	Estimated
$\frac{1}{\gamma}$	Frequency of the symptomatic infectious phase, on average	Estimated
r	proportion of symptomatic infectious disease	Estimated
S_1	Number of people who are at risk at time t_1	Estimated
E_1	quantity of individuals who were exposed at time t_1	Estimated
I_1	Number of infected people that are asymptomatic at time t_1	Estimated
N_1	Indicative infectious cases that were not identified at time t_1	Estimated
ε	Vaccine impact	0.03

Table 2: Simulations of daily reported incidents using models $AR(t)$, susceptibles $S(t)$, and cumulative reported cases $MR(t)$, $MR(t)$ since the last data point were calculated using $t = 1$ July 2022 for entirely vaccinated = 98%, 88%, 78%, and social behaviour scaling factor= 0.03, 0.023, 0.016, 0.010..

Vaccinated ε	$\varepsilon = 0.03$	$\varepsilon = 0.023$	$\varepsilon = 0.016$	$\varepsilon = 0.01$
	$AR(t) = 829$	$AR(t) = 461$	$AR(t) = 124$	$AR(t) = 25$
98%	$S(t) = 15513000$	$S(t) = 18684000$	$S(t) = 20992000$	$S(t) = 22561000$
	$MR(t) - MR(t_0) = 4513000$	$MR(t) - MR(t_0) = 3074000$	$S(t) = 20992000$	$S(t) = 22561000$
88%	$AR(t) = 3742$	$AR(t) = 1867$	$AR(t) = 589$	$AR(t) = 81$
	$S(t) = 21891000$	$S(t) = 27054000$	$S(t) = 29723000$	$S(t) = 31641000$
	$MR(t) - MR(t_0) = 4786000$	$MR(t) - MR(t_0) = 3074000$	$S(t) = 1356000$	$S(t) = 1565000$
78%	$AR(t) = 11246$	$AR(t) = 8379$	$AR(t) = 2170$	$AR(t) = 378$
	$S(t) = 26847000$	$S(t) = 37241000$	$S(t) = 41592000$	$S(t) = 38683000$
	$MR(t) - MR(t_0) = 5893000$	$MR(t) - MR(t_0) = 3936000$	$S(t) = 21320000$	$S(t) = 885000$

Acknowledgements

The authors gratefully acknowledge the approval and the support of this research study by the grand no: SCAR-2022-11-1743 from the deanship of scientific research at Northern Border University Arar, K.S.A.

References

- [1] Eid Alhmadi. Mathematical analysis of a seir model with nonlinear incidence rate for covid-19 dynamics. *Asian Research Journal of Mathematics*, pages 56–68, 2022.
- [2] Rubayyi T Alqahtani. Mathematical model of sir epidemic system (covid-19) with

- fractional derivative: stability and numerical analysis. *Advances in Difference Equations*, 2021(1):1–16, 2021.
- [3] Eitan Altman, Konstantin Avrachenkov, Francesco De Pellegrini, Rachid El-Azouzi, and Huijuan Wang. *Multilevel strategic interaction game models for complex networks*. Springer, 2019.
- [4] Fred Brauer, Carlos Castillo-Chavez, Zhilan Feng, Fred Brauer, Carlos Castillo-Chavez, and Zhilan Feng. Introduction: A prelude to mathematical epidemiology. *Mathematical Models in Epidemiology*, pages 3–19, 2019.
- [5] Bruno Buonomo. Analysis of a malaria model with mosquito host choice and bed-net control. *International Journal of Biomathematics*, 8(06):1550077, 2015.
- [6] Atsegin Canga Garcés et al. Modelling the effect of the interaction between vaccination and nonpharmaceutical measures on covid-19 incidence. 2022.
- [7] Leonardo da Vinci. Leonardo’s manuscripts. In *Handbook of Geophysical Exploration: Seismic Exploration*, volume 38, pages 443–446. Elsevier, 2007.
- [8] Luke Ekundayo Edungbola. *The Eradication of Dracunculiasis (Guinea Worm Disease) in Nigeria: An Eyewitness Account*. Academic Press, 2018.
- [9] Scott Greenhalgh and Carly Rozins. A generalized differential equation compartmental model of infectious disease transmission. *Infectious Disease Modelling*, 6:1073–1091, 2021.
- [10] MJ Keeling and P Rohani. Modeling infectious diseases in humans and animals princeton univ. *Princeton, NJ*, 2008.
- [11] Eugenio Lippiello, Giuseppe Petrillo, and Lucilla de Arcangelis. Estimating the generation interval from the incidence rate, the optimal quarantine duration and the efficiency of fast switching periodic protocols for covid-19. *Scientific Reports*, 12(1):4623, 2022.
- [12] Banan Maayah, Asma Moussaoui, Samia Bushnaq, and Omar Abu Arqub. The multi-step laplace optimized decomposition method for solving fractional-order coronavirus disease model (covid-19) via the caputo fractional approach. *Demonstratio Mathematica*, 55(1):963–977, 2022.
- [13] Maia Martcheva. *An introduction to mathematical epidemiology*, volume 61. Springer, 2015.
- [14] Edouard Mathieu, Hannah Ritchie, Esteban Ortiz-Ospina, Max Roser, Joe Hasell, Cameron Appel, Charlie Giattino, and Lucas Rodés-Guirao. A global database of covid-19 vaccinations. *Nature human behaviour*, 5(7):947–953, 2021.

- [15] Sam Moore, Edward M Hill, Louise Dyson, Michael J Tildesley, and Matt J Keeling. Modelling optimal vaccination strategy for sars-cov-2 in the uk. *PLoS computational biology*, 17(5):e1008849, 2021.
- [16] James Dickson Murray and James Dickson Murray. *Mathematical Biology: II: Spatial Models and Biomedical Applications*, volume 3. Springer, 2003.
- [17] Zaid Odibat. A universal predictor–corrector algorithm for numerical simulation of generalized fractional differential equations. *Nonlinear Dynamics*, 105(3):2363–2374, 2021.
- [18] Jørn Olsen, Kaare Christensen, Jeff Murray, Anders Ekbom, Jørn Olsen, Kaare Christensen, Jeff Murray, and Anders Ekbom. Descriptive epidemiology in clinical epidemiology. *An Introduction to Epidemiology for Health Professionals*, pages 43–47, 2010.
- [19] Arjun Puranik, Patrick J Lenahan, Eli Silvert, Michiel JM Niesen, Juan Corchadogarcia, John C O’Horo, Abinash Virk, Melanie D Swift, John Halamka, Andrew D Badley, et al. Comparison of two highly-effective mrna vaccines for covid-19 during periods of alpha and delta variant prevalence. *MedRxiv*, 2021.
- [20] Negar Bakhshi Sadabadi and Fariba Maheri. Sies epidemic model for novel covid-19 by conformable fractional derivative. 2022.
- [21] Debashis Saikia, Kalpana Bora, and Madhurjya P Bora. Covid-19 outbreak in india: an seir model-based analysis. *Nonlinear Dynamics*, 104(4):4727–4751, 2021.
- [22] Hal L Smith. *Monotone dynamical systems: an introduction to the theory of competitive and cooperative systems: an introduction to the theory of competitive and cooperative systems*. Number 41. American Mathematical Soc., 1995.
- [23] Glenn Webb. A covid-19 epidemic model predicting the effectiveness of vaccination in the us. *Infectious Disease Reports*, 13(3):654–667, 2021.
- [24] Liuqian Yu, Katja Fennel, Laurent Bertino, Mohamad El Gharamti, and Keith R Thompson. Insights on multivariate updates of physical and biogeochemical ocean variables using an ensemble kalman filter and an idealized model of upwelling. *Ocean Modelling*, 126:13–28, 2018.