Contents lists available at ScienceDirect

# MethodsX

journal homepage: www.elsevier.com/locate/methodsx

# Incidence rate drive the multiple wave in the COVID-19 pandemic

# Saroj Kumar Sahani\*, Anjali Jakhad

Faculty of Mathematics and Computer Science, Department of Mathematics, South Asian University Akbar Bhawan, Chankyapuri, New Delhi, Delhi 110021, India

#### ARTICLE INFO

Method name: Mathematical Model for COVID-19

Keywords: COVID-19 Differential equation Incidence rate MSEIR model Stability

## ABSTRACT

The last three years have been the most challenging for humanity due to the COVID-19 pandemic. The novel viral infection has eventually been able to infect most of the human population. It is now considered to be in the endemic stage, meaning it will remain in our world throughout our lifetime. There will be an intermittent outbreak of the COVID infection from time to time. Therefore, it is necessary to formulate a robust Mathematical model to study the dynamics of disease to have a control mechanism in place. In this article, we suggest a modified MSEIR model to explain the dynamics of COVID-19 infection. We assume that a susceptible person contracting the coronavirus develops a transient immunity to the illness. Further, infectives comprise asymptomatic, hospitalized and quarantined individuals. We assume that the incidence rate is of standard type, and susceptible can only become infective if they come in contact with either asymptomatic or symptomatic individuals. This basic and simple model effectively models the various waves every country has seen during the Pandemic. The simple analysis shows that the model could suggest various waves in future if we carefully select the incidence rate for the infection. In summary, we have discussed the following major points in this article.

- We have analysed for local behavior infection-free equilibrium solution. Further, a thorough
  numerical exploration with various parameter settings has been performed to obtain the different cases of infection dynamics of the coronavirus epidemic.
- We have found some interesting scenarios which explain the emergence of multiple waves observed in many countries.

#### Specifications table

Subject area:	Mathematics and Statistics
More specific subject area:	Mathematical Modelling
Name of your method:	Mathematical Model for COVID-19
Name and reference of original	The methods and techniques used are general in nature and the relevant references have been referred whenever needed
method:	
Resource availability:	Provided in the Article

#### Method details

Controlling the COVID-19 Pandemic has been a very challenging task since the year 2020 began. It is well known that to contain this pandemic, it is essential to recognize asymptomatic individuals and take appropriate steps to limit their contact with susceptible

\* Corresponding author. E-mail address: sarojkumar@sau.ac.in (S.K. Sahani).

https://doi.org/10.1016/j.mex.2023.102317 Received 5 January 2023; Accepted 3 August 2023 Available online 6 August 2023 2215-0161/© 2023 The Authors. Published by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/)







Table 1Table of state variables or compartment of model.

State Variables	Population Class
Μ	Population with Active Immunity
S	Population without Immunity
$I_1$	Asymptomatic Population
$I_2$	Symptomatic Population
H	Hospitalized Population
Q	Home Quarantined
R	Recovered Population

individuals. COVID-19 causes severe acute respiratory syndrome, whose causative agent is a novel virus known as a coronavirus. The virus is, therefore, also known as SARS-CoV-2 [1]. Generally, most of the diseases caused by the human coronavirus are mild. However, the epidemics due to two beta coronaviruses, namely SARS-CoV(severe acute respiratory syndrome coronavirus) [2–4] and MERS-CoV(Middle East respiratory syndrome coronavirus) [5–7], were somewhat lethal as they had a mortality rate of 10% and 37%, respectively. In comparison, for COVID-19 infection, the fatality rate is not as high as in other epidemics, as almost 75% of the infected individuals recover without any medication [8,9]. Also, most infected individuals do not exhibit any disease symptoms; hence they are not treated [10,11]. However, in acute cases, the recovery is difficult and may cause hospitalization; consequently, it takes 21 to 42 days to recover from the infection altogether [12].

To forecast and simulate future strategies, a mathematical model is one of the best techniques that governments and policymakers can utilize it. Mathematical model can also be used to optimize the financial and hospital resources to effectively control epidemics. A technique so-called compartmental technique in mathematical modeling are crucial mathematical tools in understanding the dynamics of epidemics. In studying these dynamics, some model parameters, such as incidence rate, mortality rate, reproduction number, infectious periods, etc., play an important role. Furthermore, using such models makes it possible to determine the so-called basic reproduction number  $R_0$ . This number may change once the disease invades the population, and the time-dependent reproduction number  $R_t$  can also be determined using these compartmental models. The time-dependent reproduction number is crucial in determining any epidemic's long-term progression.

The conventional Susceptible-Infected-Removed or simply an SIR model and its variations, including Susceptible-Exposed-Infected-Recovered-Susceptible (SEIRS), Passively Immune SEIR (MSEIR) and many more, have been widely used in the literature for epidemic modeling and prediction of disease dynamics in the past [13–15]. The conventional SIR model, put forth by Kermack-McKendrick [16], comprises a set of differential equations that use the S, I, and R compartments to describe the behavior and progression of the pandemic over some time. It has been widely used to simulate the dynamics of the infected class of individuals in the epidemics of some well-known diseases in the past. A few are the epidemics such as SARS or severe acute respiratory syndrome, MERS or middle east respiratory syndrome, influenza A etc. [17–22]. The SIR framework permits a one-way transition from infectious to susceptible. This makes sense for an infectious condition that spreads from person to person, and the recovery creates a long-lasting resistance [23]. The COVID-19 pandemic has recently been modelled using the SIR model and its variations [24–31].

In this article, we aim to develop a simple, yet effective model based on the MSEIR model to account for the various waves of infection that have evolved over the past two years.

#### Model description

We consider the human population with the total population of N. The per-capita growth of each individual is assumed to be same and equal to  $\mu$ . This assumption will lead to a constant population throughout the epidemics for a shorter time. However, we can relax this condition for some advance and future study. The population is subdivided into seven classes of individuals. Class M are the individuals having active immunity, and mostly consists of the newborn who got active immunity from their mother after birth. Second class of individuals, S are those who have lost immunity towards the disease after a certain time. We assume that whenever the individual in the compartment  $I_1$ ,  $I_2$ , Q, H or R are born, they will have some kind of active immunity in them due to their mother and hence newborn will be added to class M. However, when an individual in the S compartment is born, they will not have immunity and hence they will remain in S compartment. We consider the infected individuals who got infected with COVID-19 into two subclasses namely the asymptomatic individuals  $I_1$  and symptomatic individuals  $I_2$ . Individuals S are uninfected individuals who can become infected when they come in contact with either  $I_1$  or  $I_2$ . Since COVID-19 infections have proven to be somewhat lethal, we consider that the infected individual can either be hospitalized once their condition deteriorates or be home quarantined in case of mild symptoms. We further assume that the various class of infected individuals; namely, the asymptomatic, home quarantined and hospitalized individuals, will recover from infection after a certain time and will go to compartment R which is known as the recovered class individuals. Out of the total symptomatic individuals, we assume that a  $p_2$  fraction of them will get hospitalized and consequently go to the H compartment if their health deteriorates. Rest  $1 - p_2$  fraction will home quarantine themselves till they completely recover from the disease. The Graphical abstract gives the various compartment and the movement of an individual within the compartment. The state variables and parameters of the model are given in Table 1 and Table 2, respectively.

Table 2	
Descriptions of the model parameters.	

Parameters	Descriptions
μ	Per-Capita Birth rate of Population
d	Per-Capita Death rate of Population
β	Rate of Infection
$\delta_1$	Rate of Transfer of Individual from M class to S class
$\delta_3$	Rate of Transfer of Individual from $I_2$ class
$\delta_4$	Rate of Transfer of Individual from R class to S class
$\gamma_1$	Recovery Rate of Symptomatic Individual
$\gamma_2$	Recovery Rate from Hospitalization
γ <sub>3</sub>	Recovery Rate from of home Quarantined Individual
$p_1$	Fraction of Infected Individual who are Asymptomatic
<i>p</i> <sub>2</sub>	Fraction of Symptomatic Individual going for Hospitalization

With all the assumptions, we have the following coupled differential equation model, which describes the dynamics of COVID-19.

$$\begin{aligned} \frac{dM}{dt} &= \mu(N-S) - \delta_1 M - dM \\ \frac{dS}{dt} &= \mu S + \delta_1 M - \frac{\beta S (I_1 + I_2)}{N} - dS + \delta_3 R \\ \frac{dI_1}{dt} &= \frac{p_1 \beta S (I_1 + I_2)}{N} - \gamma_1 I_1 - d I_1 \\ \frac{dI_2}{dt} &= \frac{(1-p_1) \beta S (I_1 + I_2)}{N} - \delta_3 I_2 - d I_2 \\ \frac{dH}{dt} &= p_2 \delta_3 I_2 - \gamma_2 H - dH \\ \frac{dQ}{dt} &= (1-p_2) \delta_3 I_2 - \gamma_3 Q - dQ \\ \frac{dR}{dt} &= \gamma_1 I_1 + \gamma_2 H + \gamma_3 Q - \delta_3 R - dR \end{aligned}$$

All parameters assume to be non-negative, and the system has the initial conditions as follows:

$$M(0) \ge 0, S(0) \ge 0 \ I_1(0) \ge 0,$$
  

$$I_2(0) \ge 0H(0) \ge 0, Q(0) \ge 0, R(0)$$
(2)

#### Qualitative analysis

In this section, we present some important results for the model, which gives some basic qualitative features of the underlying model. For this, we take  $\mu = d$  and assume that the initial data satisfy  $U(0) \ge 0$ , where

 $U(t) = (M(t), S(t), I_1(t), I_2(t), H(t), Q(t), R(t))$ 

then, we can state following results for the system (1) which confirms that the model system has unique solution, solutions are positively invariant, and solutions are bounded in the solution space.

**Theorem 1.** Suppose the right side of the system (1) is  $f = [f_1, f_2, f_3, f_4, f_5, f_6, f_7]$ , then the system (1) has unique solution in the domain  $\Omega \subseteq \mathbb{R}^7$ since  $f \in C^1$  in  $\Omega$ .

**Theorem 2.** The initial conditions of the system (1) ensure that  $N(t) \ge 0$ , where  $N(t) = M(t) + S(t) + I_1(t) + I_2(t) + H(t) + Q(t) + R(t)$ . Thus, the total human population is positive and bounded for all finite time t.

**Theorem 3.** The system (1) has the bounded solution in the domain  $\Omega$  given by

$$\Omega = \{ U(t) : 0 \le U(t) \le N(0) \}$$

#### Local behavior of solution

In this section, we present the local behavior of the solution under the assumption that  $\mu = d$  which permits the equilibrium points of the system. The system (1) has two types of equilibrium points, namely:

1. The disease-free equilibrium state  $J_0 = (0, N, 0, 0, 0, 0, 0)$ 

(1)

(3)

2. the infected equilibrium state  $J^* = (M^*, S^*, I_1^*, I_2^*, H^*, Q^*, R^*)$ 

where *N* is the total population which is constant for  $\mu = d$ . The Jacobian Matrix evaluated at  $J_0$  is

$$\begin{bmatrix} -d-\delta_1 & -d & 0 & 0 & 0 & 0 & 0 \\ \delta_1 & 0 & -\beta & -\beta & 0 & 0 & \delta_4 \\ 0 & 0 & -d+p_1\beta-\gamma_1 & p_1\beta & 0 & 0 & 0 \\ 0 & 0 & (1-p_1)\beta & -d+(1-p_1)\beta-\delta_3 & 0 & 0 & 0 \\ 0 & 0 & 0 & p_2\delta_3 & -d-\gamma_2 & 0 & 0 \\ 0 & 0 & 0 & (1-p_2)\delta_3 & 0 & -d-\gamma_3 & 0 \\ 0 & 0 & \gamma_1 & 0 & \gamma_2 & \gamma_3 & -d-\delta_4 \end{bmatrix}$$

The characteristic equation for the Jacobian Matrix is given by  $|J_0 - \lambda I| = 0$  which is

$$(\lambda + d)(\lambda + \delta_1)(\lambda + d + \delta_4)(\lambda + d + \gamma_2)(\lambda + d + \gamma_3)(\lambda^2 + \lambda C_1 + C_2) = 0$$

The eigenvalues of  $J_0$  are -d,  $\delta_1$ ,  $-d - \delta_4$ ,  $-d - \gamma_2$ ,  $-d - \gamma_3$  and other two determined from the quadratic

$$\lambda^2 + \lambda C_1 + C_2 = 0$$

where

 $C_1 = -\beta + \gamma_1 + 2 d + \delta_3$ 

$$C_2 = -\beta \gamma_1 + \gamma_1 \delta_3 + d^2 - \beta d + \gamma_1 d + \delta_3 d + \beta \gamma_1 p_1 - \beta \delta_3 p_1.$$

Hence the conditions for local stability conditions for  $J_0$  are given by the following theorem.

**Theorem 4.** The system (1) has disease-free equilibrium point  $J_0$ , which is stable if any one of the conditions is satisfied

$$\begin{aligned} \mathbf{S1} & 0 < \beta \le d \\ \mathbf{S2} & d < \beta < \gamma + d, \ \delta_3 < \beta - d, \ \frac{\beta \gamma_1 - \gamma_1 \delta_3 - d^2 + \beta \ d - \gamma_1 \ d - d \ \delta_3}{\beta \gamma_1 - \beta \ \delta_3} < p_1 < 1 \\ \mathbf{S3} & d < \beta < \gamma_1 + d, \ \delta_3 = \beta - d \\ \mathbf{S4} & d < \beta < \delta_3 + d, \gamma_1 < \beta - d, \ 0 < p_1 < \frac{\beta \gamma_1 - \gamma_1 \delta_3 - d^2 + \beta \ d - \gamma_1 \ d - d \ \delta_3}{\beta \gamma_1 - \beta \ \delta_3} \\ \mathbf{S5} & d < \beta \langle \gamma_1 + d, \delta_3 \rangle \beta - d \end{aligned}$$

For the endemic equilibrium point, the complexity of the model does not allow us to do a local stability analysis of the model. Hence, we leave this analysis to a future study. We now perform a exhaustive numerical long-term dynamic simulations of the model for various case study.

#### Numerical simulation with case studies

In order to have a more general model, we allow the incidence rate coefficient  $\beta$  to vary with respect to time to account for the variable transmission rate of the disease. We, therefore, assume that  $\beta = \beta(t)$ .

The more logical assumption for this type of function is a periodic function which also accounts for seasonal variation in the infectivity of the virus due to some natural factors. We also generalize model (1) with the introduction of some more parameters into the system to get the following set of dynamic equations as follows.

$$\begin{aligned} \frac{dM}{dt} &= \mu(N-S) - \delta_1 M - dM \\ \frac{dS}{dt} &= \mu S + \delta_1 M - \frac{\beta S (I_1 + I_2)}{N} - dS + \delta_4 R \\ \frac{dI_1}{dt} &= \frac{p_1 \beta S (I_1 + I_2)}{N} - \gamma_1 I_1 - d \ I_1 - \sigma_1 \ I_1 - \delta_2 I_1 \\ \frac{dI_2}{dt} &= \frac{(1-p_1)\beta S (I_1 + I_2)}{N} - \delta_3 I_2 - d \ I_2 - \sigma_2 \ I_2 + \delta_2 I_1 \\ \frac{dH}{dt} &= p_2 \delta_3 I_2 - \gamma_2 H - dH - \sigma_3 H \\ \frac{dQ}{dt} &= (1-p_2) \delta_3 I_2 - \gamma_3 Q - dQ - \sigma_4 \ Q \\ \frac{dR}{dt} &= \gamma_1 I_1 + \gamma_2 H + \gamma_3 Q - \delta_4 R - dR \end{aligned}$$

Here the newly introduced parameters  $\sigma_1$ ,  $\sigma_2$ ,  $\sigma_3$  and  $\sigma_4$  are extra death rates due to COVID infection in asymptomatic, symptomatic, hospitalized and home quarantined individuals, respectively. These assumptions are well in line with the actual scenario where



Fig. 1. Plot showing population of individuals in various compartment for Case 1.

we have observed that death varies among different classes of individuals. Moreover, we assume that there could be a transfer of individuals from asymptomatic to symptomatic class with the rate  $\delta_2$ .

#### Case 1. Single Wave

In this case, we solve our original system of Eq. (3) with constant  $\beta$ . We assume the following set of parameter values to numerically solve the underlying system

$$d = 0.00214, \mu = 0.002, \delta_1 = 0.09,$$
  

$$\delta_2 = 0.0752, \delta_3 = 0.06, \ \delta_4 = 0.01, \gamma_1 = 0.16,$$
  

$$\gamma_2 = 0.9, \gamma_3 = 0.07, \sigma_1 = 0.0484, \sigma_2 = 0.06$$
  

$$\sigma_3 = 0.09, \sigma_4 = 0.022778, \ p_1 = 0.5, \ p_2 = 0.4$$
(4)

The case is depicted in Fig. 1(a)–(d) for different values of  $\beta$ . It can be observed from these figures that there can be only one wave of infection possible in the epidemic. The disease ultimately stabilizes, or the infection dies out in the longer run. There can never be a pandemic, and hence the constant value of  $\beta$  cannot explain the occurrence of multiple waves in the COVID-19 epidemic. The lesser value of  $\beta$  also flattens the infection curve, or the higher value of  $\beta$  gives steeper wave, as evident.

#### Case 2. Multiple Waves with 1st Wave having large spike

For this case, we solve our original system of Eq. (3) with a variable  $\beta(t)$ . We take the following parameter values

$$d = 0.00714, \mu = 0.008, \delta_1 = 0.09,$$
  

$$\delta_2 = 0.0752, \delta_3 = 0.06, \ \delta_4 = 0.01, \gamma_1 = 1.0,$$
  

$$\gamma_2 = 1.0, \gamma_3 = 0.07, \sigma_1 = 0.0484, \sigma_2 = 0.06$$
  

$$\sigma_3 = 0.09, \sigma_4 = 0.022778, \ p_1 = 0.6, \ p_2 = 0.1$$
(5)

This case gives rise to multiple waves in the epidemic, with the first wave infecting more persons than the latter. The lower amplitude wave for the latter one can be due to the lower infectiousness of the virus due to the development of immunity against the virus, possibly because of the attainment of herd immunity in the first wave. The case is depicted in Fig. 2(a)–(d). This shows that COVID-19 can take the form of an epidemic in the population, and infection will remain in the population for a lifetime.



Fig. 2. Plot showing population of individuals in various compartment for Case 2.



Fig. 3. Plot showing population of individuals in various compartment for Case 3.



Fig. 4. Plot showing population of individuals in various compartment for Case 4.



Fig. 5. Plot showing population of individuals in various compartment for Case 5.



Fig. 6. Plot showing population of individuals in various compartment for Case 5.

#### Case 3. Multiple Waves with 2nd Wave having large spike

Again, in this case, we solve our original system of Eq. (3) with parameter values as given in set (5) with a variable  $\beta(t)$ , a function of time *t*.

In this scenario, too, the model gives rise to multiple waves in the epidemic, with the second wave infecting more persons than the other one. The lower amplitude wave for the latter one can be due to the lower infectiousness of the virus due to the development of immunity against the virus, possibly because of the attainment of herd immunity in the first two waves. The case is depicted in Fig. 3(a)–(d). This again shows that COVID-19 can take the form of an epidemic in the population, and infection will remain in the population for lifetime.

#### Case 4. Multiple Waves with 2nd and 3rd Waves having large spike

This case can again be obtained from the solution of the system of Eq. (3) with parameter values as given in set (5) with a variable  $\beta(t)$ .

In this scenario, the system gives rise to multiple waves in the epidemic, with the second wave and third wave infecting more persons than the other one. This is a more practical situation of COVID infection in some countries where there have been multiple waves, with 2nd wave proving to be more infectious. The case is depicted in Fig. 4(a)-(d).

#### Case 5. Multiple Waves with 2nd and 3rd Waves having large spike

This case is also obtained by solving the system of Eq. (3) with parameter values as given in set 5 with a variable  $\beta(t)$ .

In this scenario, the system gives rise to multiple waves in an epidemic, with the second wave having a large spike than the others. The other wave's spike reduces as time progress. In one of the cases, we can see no appreciable spike after the third wave, and it appears that the disease dies out completely (see Fig. 5(a)-(d)), which can be a rare case. Another more logical case is the one where disease prevails in the population, but the spike in the wave diminishes as time passes out, which can be seen in Fig. 6(a)-(d).

#### Conclusion

In this article, we proposed one modified MSEIR model considering the temporary immunity that the coronavirus imparts to infected individuals with four types of subclasses of infected individuals: asymptomatic, symptomatic, hospitalized, and homequarantined individuals. In this model, we assumed that the infection rate could be variable, which accounts for the emergence of various waves during the COVID-19 pandemic. We have numerically simulated the model with a different set of parameter values and found a variety of infection dynamics possible from this simple model. We can effectively explain the various waves observed in the coronavirus epidemic. With the application of this model, we can now develop a control model that can be applied to this model which controls the infection.

In this study, we presented a modified MSEIR model for the COVID-19 pandemic and illustrated various theoretical cases which may lead to various types of waves of infection. Although the model's outcome is in good agreement with the infection dynamics in some countries, to validate it correctly, we need to present a model validation through some data. In our future study, we are working on validating this model with actual COVID data. We are in the process of collecting the data of various classes of individuals and trying to estimate the parameters involved in this article. We will also work on the controlling parameter that can curtail the long-term epidemic.

### **Ethics statements**

Nil.

### **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.

#### CRediT authorship contribution statement

Saroj Kumar Sahani: Conceptualization, Methodology, Supervision, Software, Writing – original draft. Anjali Jakhad: Validation, Visualization, Investigation, Writing – review & editing.

#### Data availability

No data was used for the research described in the article.

#### Acknowledgments

The second author is supported by "SAARC Silver Jubilee Scholarship" from South Asian University, New Delhi, India to carry out this research.

#### References

- [1] W.H. Organization, Naming the coronavirus disease (COVID-19) and the virus that causes it, Braz. J. Implantol. Health Sci. 2 (3) (2020).
- [2] T.G. Ksiazek, D. Erdman, C.S. Goldsmith, S.R. Zaki, T. Peret, S. Emery, S. Tong, C. Urbani, J.A. Comer, W. Lim, et al., A novel coronavirus associated with severe acute respiratory syndrome, N. Engl. J. Med. 348 (20) (2003) 1953–1966.
- [3] T. Kuiken, R.A. Fouchier, M. Schutten, G.F. Rimmelzwaan, G. Van Amerongen, D. Van Riel, J.D. Laman, T. De Jong, G. Van Door- num, W. Lim, et al., Newly discovered coronavirus as the primary cause of severe acute respiratory syndrome, Lancet N. Am. Ed. 362 (9380) (2003) 263–270.
- [4] C. Drosten, S.G. ünther, W. Preiser, S. Van Der Werf, H.R. Brodt, S. Becker, H. Rabenau, M. Panning, L. Kolesnikova, R.A. Fouchier, et al., Identification of a novel coronavirus in patients with severe acute respiratory syn- drome, N. Engl. J. Med. 348 (20) (2003) 1967–1976.
- [5] D.K. Bonilla-Aldana, K. Quintero-Rada, J.P. Montoya-Posada, S. Ramírez-Ocampo, A. Paniz-Mondolfi, A.A. Rabaan, R. Sah, A.J. Rodríguez-Morales, SARS-COV, MERS-COV and now the 2019-novel cov: have we investigated enough about coronaviruses?-a bibliometric analysis, Travel Med. Infect. Dis. 33 (2020) 101566.
- [6] J.A. Al-Tawfiq, Asymptomatic coronavirus infection: MERS-COV and SARS- COV-2 (COVID-19), Travel Med. Infect. Dis. 35 (2020) 101608.
- [7] A. Chafekar, B.C. Fielding, Mers-cov: understanding the latest human coronavirus threat, Viruses 10 (2) (2018) 93.
- [8] O. Albitar, R. Ballouze, J.P. Ooi, S.M.S. Ghadzi, Risk factors for mortality among COVID-19 patients, Diabetes Res. Clin. Pract. 166 (2020) 108293.
- [9] G. Sousa, T. Garces, V. Cestari, R. Florêncio, T. Moreira, M. Pereira, Mortality and survival of COVID-19, Epidemiol. Infect. 148 (2020) e123, doi:10.1017/S0950268820001405.
- [10] B. Ivorra, M. Ferrández, M. Vela-Pérez, A. Ramos, Mathematical modeling of the spread of the coronavirus disease 2019 (COVID-19) considering its particular characteristics. the case of China, Commun. Non-linear Sci. Numer. Simul. 88 (2020) 105303.
- [11] N. Al-Awwal, F. Dweik, S. Mahdi, M. El-Dweik, S.H. Anderson, A review of SARS-COV-2 disease (COVID-19): pandemic in our time, Pathogens 11 (3) (2022) 368.
- [12] C. Gomes, Report of the who-China joint mission on coronavirus disease 2019 (COVID-19), Braz. J. Implantol. Health Sci. 2 (3) (2020).
- [13] H.W. Hethcote, The mathematics of infectious diseases, SIAM Rev. 42 (4) (2000) 599-653.
- [14] F. Ndaïrou, I. Area, J.J. Nieto, D.F. Torres, Mathematical modeling of COVID-19 transmission dynamics with a case study of wuhan, Chaos Soli- tons Fractals 135 (2020) 109846.
- [15] X. Chen, A. Zhang, H. Wang, A. Gallaher, X. Zhu, Compliance and containment in social distancing: mathematical modeling of COVID-19 across townships, Int. J. Geogr. Inf. Sci. 35 (3) (2021) 446–465.
- [16] W.O. Kermack, A.G. McKendrick, Contributions to the mathematical theory of epidemics-i. 1927, Bull. Math. Biol. 53 (1-2) (1991) 33-55.
- [17] E.J. Schwartz, B. Choi, G.A. Rempala, Estimating epidemic parameters: application to H1N1 pandemic data, Math. Biosci. 270 (2015) 198-203.
- [18] L. Laguzet, G. Turinici, Individual vaccination as nash equilibrium in a sir model with application to the 2009–2010 influenza a (H1N1) epidemic in France, Bull. Math. Biol. 77 (10) (2015) 1955–1984.
- [19] X. Huang, A.C. Clements, G. Williams, K. Mengersen, S. Tong, W. Hu, Bayesian estimation of the dynamics of pandemic (H1N1) 2009 influenza transmission in Queensland: a space-time SIR-based model, Environ. Res. 146 (2016) 308–314.
- [20] T. Mkhatshwa, A. Mummert, Modeling super-spreading events for infectious diseases: case study SARS, arXiv preprint (2010) arXiv preprint arXiv:1007.0908.
- [21] J.O. Giraldo, D.H. Palacio, Deterministic SIR (susceptible-infected- removed) models applied to varicella outbreaks, Epidemiol. Infect. 136 (5) (2008) 679–687.
   [22] B. Yong, L. Owen, Dynamical transmission model of MERS-COV in two areas, AIP Conf. Proc. 1716 (2016) 020010.
- [23] J.C. Blackwood, L.M. Childs, An introduction to compartmental modeling for the budding infectious disease modeler, Lett. Biomath. 5 (1) (2018) 195–221.
- [24] C. You, Y. Deng, W. Hu, J. Sun, Q. Lin, F. Zhou, C.H. Pang, Y. Zhang, Z. Chen, X.H. Zhou, Estimation of the time-varying reproduction number of COVID-19 outbreak in China, Int. J. Hyg. Environ. Health 228 (2020) 113555.

- [25] F. Najafi, N. Izadi, S.S. Hashemi-Nazari, F. Khosravi-Shadmani, R. Nikbakht, E. Shakiba, Serial interval and time-varying reproduction number estimation for COVID-19 in western Iran, New Microbes New Infect. 36 (2020) 100715.
- [26] C.E. Wagner, C.M. Saad-Roy, B.T. Grenfell, Modelling vaccination strategies for COVID-19, Nat. Rev. Immunol. 22 (3) (2022) 139–141.
- [27] M.S. Alqarni, M. Alghamdi, T. Muhammad, A.S. Alshomrani, M.A. Khan, Mathematical modeling for novel coronavirus (COVID-19) and control, Numer. Methods Partial Differ. Eqs. 38 (4) (2022) 760-776.
- [28] K. Liu, Y. Lou, Optimizing COVID-19 vaccination programs during vaccine shortages: a review of mathematical models, Infect. Dis. Model. 7 (1) (2022) 286–298.
   [29] E. Antonelli, E.L. Piccolomini, F. Zama, Switched forced SEIRDV compartmental models to monitor COVID-19 spread and immunization in Italy, Infect. Dis. Model. 7 (1) (2022) 1-15.
- [30] A. Adiga, D. Dubhashi, B. Lewis, M. Marathe, S. Venkatramanan, A. Vullikanti, Mathematical models for COVID-19 pandemic: a comparative analysis, J. Indian Inst. Sci. 100 (4) (2020) 793-807.
- [31] A. Leontitsis, A. Senok, A. Alsheikh-Ali, Y. Al Nasser, T. Loney, A. Alshamsi, Seahir: a specialized compartmental model for COVID-19, Int. J. Environ. Res. Public Health 18 (5) (2021) 2667.