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Infectious disease epidemiology under meteorological factors: A review of mathematical models and an extended SEIR framework

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Received: 19 March 2025, Published: 24 June 2025

Abstract: **Mathematical modeling can perform a decisive task in understanding, controlling, and preventing the transmission of infectious diseases by forecasting their spread, estimating the effectiveness of intervention measures, and updating public health policies. A mathematical epidemic model is a vital tool that can mock up the spread of infections under different scenarios and environments, allowing researchers to test and refine their understanding of the fundamental mechanisms. This paper attempts to review some existing mathematical compartmental epidemic models and explore the impact of meteorological factors such as air temperature, humidity, and wind speed on epidemiology. The goal is to identify and categorize key components, research trends, major findings, and gaps within the models. Additionally, the paper discusses some strategies to address these gaps and proposes a compartmental augmentation of the SEIR model incorporating meteorological factors for further work.**

Keywords: **infectious diseases, compartmental model, transmission dynamics, basic reproduction number, meteorological factors**

I. Introduction

Infectious diseases are health problems initiated by harmful microbes viz. viruses, bacteria, fungi and parasites that enter, multiply and cause various symptoms to the living body. These pathogens can spread from host to host through direct contact, airborne route, a vector, droplets, contaminated food and infected surfaces. Infections induced by bacteria, namely streptococcus, staphylococcus and E. Coli etc. consist of strep throat, boil and urinary tract infections respectively. Viral infections, triggered by viruses like adenovirus, influenza virus, rhinoviruses, coronavirus and retrovirus include conjunctivitis, flu, common cold, COVID-19 and AIDS respectively. Lice, tapeworms and malaria are parasitic infections. Considerable effects of infectious diseases on individuals, communities and societies can comprise social effects, economic effects, psychological effects etc [1]. Their outbreaks can display social stigma and

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Citation: Shiva Hari Subedi, Russell Alpizar-Jara, Gyan Bahadur Thapa, Infectious disease epidemiology under meteorological factors: A review of mathematical models and an extended SEIR framework, Biomath 14 (2025), 2506245, https://doi.org/10.55630/j.biomath.2025.06.245

discrimination towards infected individuals, as well as quarantine and travel restrictions disordering normal social interactions and activities [2]. Epidemics can result in crucial economic costs, including direct costs for healthcare and indirect costs due to loss of productivity as well as disruptions to trade and commerce. They have the potential to cause PTSD (post-traumatic stress disorder), dread, loneliness, sadness, and anxiety [3].

In recent years, there has been growing attention to combining meteorological factors into epidemic models to understand and forecast disease transmission. Meteorological components like temperature, humidity, air currents and air pollution can affect the survival and transmission of infectious agents. For example, certain viruses, like influenza, tend to spread more efficiently in colder and drier conditions [4], while others, like some corona-viruses, may exhibit seasonal variations based on temperature and humidity [5]. It is strongly believed that viral respiratory infections trigger in winter around temperate climate regions [6], and ambient temperature and humidity take part in the seasonal characters of their occurrences [7, 8]. Studies have exposed that low relative or absolute humidity and low air temperatures increase the survival and transmission of influenza viruses [7, 9].

Generally, low outdoor temperature, depleted humidity and small wind speed (mainly in summer) are seen to be associated with increased COVID-19 transmission [10, 11]. Measles is highly contagious and spreads through the air [12]; however, higher temperature, humidity, and wind speeds generally reduce transmission [13, 14]. A study conducted during the SARS outbreak concluded that low relative humidity in low air temperatures causes a rise in viral transmission [15]. Tuberculosis is also transmitted through the air and meteorological conditions such as humidity, air temperature can inversely affect the transmission of the bacteria [16]. It has been observed that cities with mild winters and warm summers have amplified number of dengue emergence [17] and it correlates positively with relative humidity and average minimum temperature but adversely correlated with rainfall, temperature within the same period, and wind speed [18].

Mathematical modeling of infectious diseases in diverse meteorological conditions is an important part of disease-related research as epidemics/pandemics continue to be a significant issue for public health worldwide. Besides meteorological factors, some cultural practices, social norms and economic inequalities can play an expressive role in disease transmission [19]. The fundamental ideas of mathematical modeling of infectious diseases in diverse meteorological conditions include the transmission dynamics, the meteorological factors, and the efficiency of control measures in these conditions. It is based on several theories of infectious disease dynamics, population dynamics, mass action principle and environmental health, etc. However, several research gaps in related research indicate the demand for more accurate meteorological and disease transmission data, the formulation of more realistic models that capture the complexity of transmission and the evaluation of valuable control measures in different environments. The use of more sophisticated models, network and individual-based models as well as the advancements in data collection and analysis have facilitated the development of more accurate models and predictions.

The structure of the remaining part of the review paper is as follows: Section II presents some terminologies of epidemic models followed by some background of the study in Section III, theoretical framework of different epidemic compartmental models in Section IV, discussion with research gaps in Section V, and some applications in Section VI. Finally, Section VII concludes our review.

II. Terminologies

This section provides definitions and explanations of key terminologies used in mathematical modeling within infectious disease epidemiology.

A. Epidemiology

Epidemiology is a branch of health discipline that surveys epidemics with their influencing factors, spreading patterns, and controlling mechanisms in human populations. Identifying the roots and risk issues linked with specific diseases, epidemiology delivers the tools and methods needed to monitor, minimize and rheostat the epidemics/pandemics. Epidemiological studies are mainly based on frequencies, patterns, trends, risk factors, control measures, population, ecology, lab tests, genetic factors and transmission dynamics. According to the diseases, epidemiology can be classified into infectious diseases epidemiology, chronic diseases epidemiology, environmental epidemiology, violence and injury epidemiology, etc. Infectious disease epidemiology helps in detecting pathogens, understanding the patterns and causes of their spread, and devising effective interventions for defensive control. The procedures of epidemiological studies mainly include descriptive, experimental and analytic techniques. Descriptive epidemiology describes the distribution of

Notation	Definition	Unit
$S = S(t)$	Susceptible individuals	
$E = E(t)$	Exposed individuals	
$I = I(t)$	Infected individuals	
$I_s(t)$	Symptomatic	
$I_a(t)$	Asymptomatic infected	
$H_m = H_m(t)$	Home isolated	$individuals$
$Q = Q(t)$	Quarantined	
$H = H(t)$	Hospitalized	
$D = D(t)$	Death	
$R_c = R_c(t)$	Recovered	
$R = R(t)$	Removed $(R_c + D)$	

Table 1: Epidemiological compartments

N.	Meaning	Unit
α	Birth rate	
ω	Natural death rate	
δ	Infection-induced average fatality rate	
β	Transmission rate: Probability of diseases transmission/contact $\times$ no. of contacts/day	per day
ρ	Recovery rate	
ϵ	Infection rate from exposed to infectious	
ν	Immunity loss rate	
$1/\epsilon$	Average incubation period	
$1/\nu$	Infectious period	$days$
$\mathcal{R}_0$	Basic reproduction number	$ratio$

Table 2: Epidemiological parameters

disease in a population whereas analytic epidemiology examines the relationships between exposure to specific risk factors and the occurrence of disease. Experimental epidemiology tests interventions to prevent or treat disease in controlled settings. Ecological epidemiology studies the relationship between disease and environmental or lifestyle factors. Mathematical or statistical epidemiology uses mathematical models (expressed as differential or difference equations) and statistical methods (in terms of stochastic formulation) to study the transmission of disease and to calculate the result of interventions on disease control. There are several types of models based on the nature and time interval of the diseases, population size, the purpose of the study, availability of data, resources, etc. Some of the major examples consist of compartmental, deterministic, stochastic, agent-based, network and spatial models.

B. Epidemiological compartments and parameters

Epidemiological compartments are the partitions of the population $N = N(t)$ at a time t, categorized to separate individuals based on their disease status during an epidemic or a pandemic. Epidemic parameters are quantitative metrics used in epidemiology to describe and quantify the characteristics of diseases within a population. The compartments of the total population $N = N(t)$ used in the models are summarized in Table 1 and the related parameters (except for the proposed model in Section IV-F) are presented in Table 2.

C. Basic reproduction number

The basic reproduction number, symbolized by $\mathcal{R}_0$, is a key epidemic parameter, which counts the number of mean secondary infections produced by one infected person in a fully susceptible population (all individuals are not immunized naturally or through vaccination). If $\mathcal{R}_0 < 1$, the disease will vanish, while if $\mathcal{R}_0 > 1$, the illness will progress and potentially cause an epidemic, and $\mathcal{R}_0 = 1$ means the cases are stable over time. The value of $\mathcal{R}_0$ mainly depends upon the infectious period, probability of infection being transmitted during interaction among infected and susceptible persons, and average rate of contact between them. The increment in transmission rate (β) causes increase in $\mathcal{R}_0$ whereas recovery rate (ρ), and immunity loss rate (ν) are inversely proportional to $\mathcal{R}_0$ under the various epidemic model. Some challenges in determining the value of $\mathcal{R}_0$ arises because it is an average and there may be superspreaders among the population [20]. Mutations of pathogens and asymptomatic spreading also complicate its estimation [21, 22].

D. Deterministic compartmental model

A deterministic compartmental model is a mathematical simulation applied in epidemiology to explore the spread of infectious diseases by dividing a population into several compartments based on their disease category. This uses deterministic equations to describe the movement of individuals among compartments over time. The basic structure of this model includes a system of ordinary differential equations that describes the differentiation of the number of entities with respect to time in each compartment. The model considers various factors such as the rates of disease transmission, recovery and mortality, providing a baseline representation of spread of the diseases. The models are used to determine $\mathcal{R}_0$ of a disease, which is a measure of its transmission potential, to predict the upcoming spread of disease and to evaluate the impression of interventions, for instance, vaccination and isolation. Some compartmental models that can be formulated with a deterministic approach include SI, SIS, SIR, SEIR, SEIRS etc. However, deterministic compartmental models have some limitations, as they are not able to reflect random fluctuations in disease transmission that occur in reality [23]. To address these

limitations, stochastic models are often used in conjunction with deterministic models to provide a more realistic representation of the transmission of infections.

III. Background

The first epidemic model is generally attributed to D. Bernoulli [24], who proposed a mathematical version in 1766 for the spread of smallpox explaining the outcomes of smallpox variolation (a predecessor of vaccination). Classifying a population into two clusters: susceptible and immune, he proposed that the likelihood of contracting smallpox is proportional to the number of susceptible individuals. More complex models were developed later, but Bernoulli's model remains a useful starting point for understanding epidemic dynamics. In the 19th century, Farr [25] prepared a report on London's cholera epidemic analyzing mortality rates and other health-related data and the report contributed to understanding the transmission of cholera and to determining the laws governing the rise and fall of epidemics.

The deterministic approach in the epidemic model was launched at the beginning of the 20th century. Hamer [26] expressed a model with discrete time to identify the reappearance of measles epidemics and applied the model to investigate the role of vaccination and isolation. He projected that the quantity of infectious and vulnerable people determines how far the disease can spread, and this indication has been fundamental in compartmental models. Ross [27] designed a system of differential equations as a host-vector disease model with the concept of the basic reproduction number, providing a straightforward compartmental model including humans and mosquitoes. Field trials defended this supposition and directed to a brilliant achievements in malaria control [28]. After that many researchers contributed to deterministic epidemiology modeling [29]. Bernoulli's model was later refined by other mathematicians, including Kermack et al. [30] who boosted the exponential growth in mathematical epidemiology developing a collection of differential equations that could be applied to model infectious diseases dynamics. Their classic SIR model assumes that all individuals are initially equally susceptible, the disease is transmitted through direct contact, and individuals gain complete immunity after recovery. As this model was assumed to be sufficient to explain a simple epidemic but not enough for endemic disease transmission, they extended their theory to tolerate for birth, migration, death and imperfect immunity [31,32].

Soper [33] was the author to suggest the seasonality of meteorological conditions and the school calendar as deterministic phenomena in epidemiological time series. His review article discussed the statistical analysis of periodicity in disease prevalence producing damped oscillations in contrast to the observations. Brownlee [34] presented a Fourier analysis of the time series of deaths by measles epidemics and investigated the cyclicity of the epidemics using the periodogram method. London and Yorke [35] investigated the seasonal variation in contact rates for recurrent epidemics of chickenpox , measles and mumps by formulating ordinary differential delay equations. They obtained undamped oscillations with periods of one and two years using a deterministic model with a latent period. Stirzaker [36] considered stochastic approaches of interrelating populations, particularly for the development of a recurrent epidemic in huge population, concluding seasonal effects cause periodic fluctuations in contact and immigration rate. Dietz [37] presented a mathematical model with different compartments to analyze the occurrence of infectious diseases with vaccinations, latent period and seasonal variations of the contact rate. An SEIR model with sub-harmonic bifurcation, well documenting the presence of periodic and non-periodic performance in recurring epidemics was analyzed [38].

Grassly and Fraser [39] investigated the causes and effects of seasonality and came to a number of novel conclusions about vaccination policy and disease outbreak interpretation. They discussed the implications of seasonality for $\mathcal{R}_0$, as well as the mathematical analysis of routine and pulse immunization programs against seasonal illnesses, disease outbreaks, and endemic dynamics. Acari et al. [40] carried out a statistical analysis on the variations among regions in dengue/DHF and their climatic relationships. They found that a variety of additional elements unique to the local surroundings also seem to be involved, even while climatic factors show a significant act in shaping the timing and intensity of the outbreaks. Yi et al. [41] examined the dynamical behavior of an SEIR model with seasonal forcing in the transmission rate by using algebraic and differential equations, considering different number of parameters as variables. Shaman et al. [9] statistically reanalyzed previous studies on effect of relative humidity on the transmission of influenza virus and their survival. They reported that low relative or absolute humidity increases the transmission and survival of the viruses. Chan et al. [15] conducted an experimental study in Hong Kong during the 2003 outbreak of SARS-CoV-1 and remarked that low relative humidity and low temperature are

associated with increased transmission of the virus. Yang et al. [42] performed a study focusing on the impact of relative humidity on the viruses of airborne diseases and reported that many airborne viruses are seemed to be sensitive to ambient humidity affecting the capability of aerosol's viruses.

Lowen and Steel [7] used an SEIR model to investigate the impact of humidity and temperature on influenza transmission. They indicated that higher temperatures and lower humidities are associated with higher transmission rates. Abdelrazec and Gumel [43] designed a compartmental deterministic nonlinear differential equation model to study the impact of the temperature and rainfall on mosquito population and identified a range of temperature and rainfall suitable for their growth. Naserpor et al. [44] formulated, exploited and compared three variations of SIR model with climate data, one of them being modification obtained by multiplying a linear combination of precipitation, humidity and temperature by the spread rate. They found that precipitation had the highest link with the dynamics of influenza transmission out of the three environmental parameters. Arquam et al. [45] investigated an improved SIR model for spread of vector-borne diseases, considering host and vector populations, to assess the impact of temperature. They discovered that the square of biting rate, depending on temperature determines the threshold of the transmission rate. They also came to the conclusion that the outbreak would end if the temperature rose over $37°C$ because the vector population would go extinct. Islam et al. [10] analyzed the data from 116 countries that reported confirmed cases of COVID-19 and found that low temperature, low humidity, and low wind speed are associated with high transmission. Mecenas et al. [46] conducted a review on the effect of temperature and humidity on COVID-19 transmission, finally selecting eligible 17 articles from an initial screening of 517 articles related to their concerns. They observed great homogeneity in the findings regarding their research problem and put a conclusion that hot and humid environment reduce the transmission.

Clouston [47] used a statistical model to investigate the effects of wind speed on the transmission of SARS-CoV-2 and recommended that higher wind speeds cause lower transmission rates. Verheyen et al. [48] arranged the COVID-19 data with meteorological measurements for 121 countries and reported that maintaining an indoor RH in $40\% - 60\%$ is linked with comparatively decreased infection and mortality rates, while situations outside this range are associated with worse outcomes. Bozic et al. [49] explained the physical mechanisms of respiratory transmission of infectious diseases through air and droplet influenced by RH, and made a conclusion that interior $RH \approx 50\%$ is highly appropriate for human comfort and possibly a right target to support in controlling the spread of the infections. Coccia [50] conducted a statistical analysis of the impact of environmental pollution and climatological factors on COVID-19. The study found that low air current with high pollution can contribute significantly to the spread of COVID-19 and other infectious diseases, particularly during the fall and winter seasons. Yuan et al. [11] used Distributed Lag Nonlinear Model and Generalized Additive Models to investigate the relationship between new COVID-19 cases and global climatic conditions for the entire year 2020. Their research showed that while the daytime range of temperatures is favorably connected with new cases every day, the average temperature, speed of wind, and relative humidity are adversely associated. Wu et al. [51] examined the relationship between temperature and relative humidity with daily COVID-19 new cases and fatalities in 166 countries. They found that these variables were adversely correlated. Nuraini et al. [52] used auto-regressive distributed lag (ARDL) method to model the transmission dynamic of dengue denoting the rate of infection as a function of climate variables. They stated that while humidity and precipitation are positively correlated with illnesses, the ideal temperature for infection ranges between 24.3°C and 30.5°C. Li et al. [53] adopted improved SEIR model and used Pearson correlation to investigate the connection between the environment and the COVID-19 outbreak.

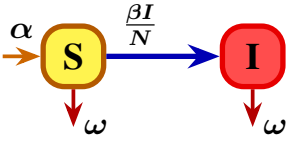
IV. Mathematical models of epidemics

This section presents theoretical framework of some familiar deterministic compartmental epidemic models along with a proposed model for airborne diseases.

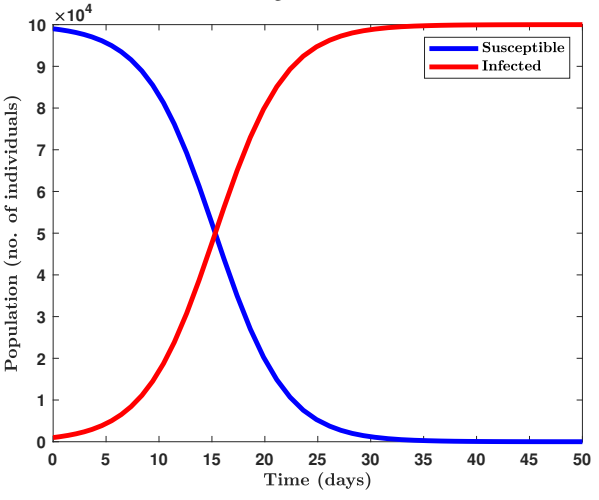
A. Susceptible-Infected (SI) model

SI model is the basic compartmental epidemic model which assumes that individuals born without immunity, if infected and receive no medication, always become infected and contagious because of continued interaction with the susceptible people. The basic SI model is suitable for the dynamical analysis of the diseases like cytomegalovirus (CMV) or herpes.

The foundation of this model develops the supposition that the likelihood of a susceptible person becoming infected by a contagious person is directly proportional to the total figure of contagious persons.

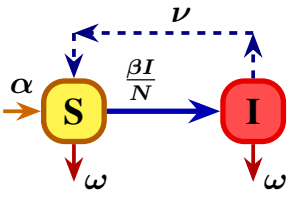


(A) Flow diagram of SI model.

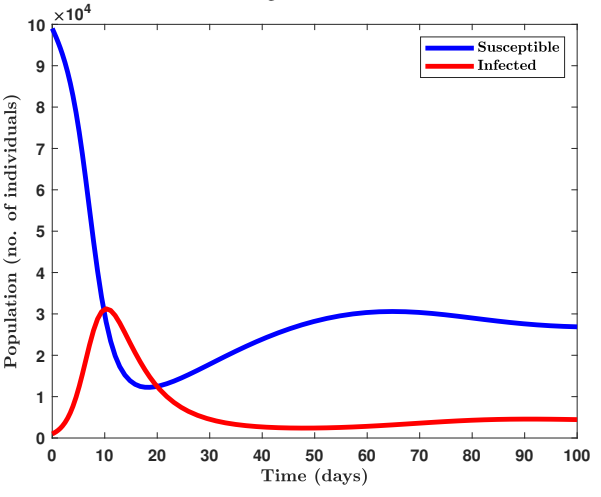


(B) Solution of SI model without vital dynamics.

Fig. 1: SI model.



(A) Flow diagram of SIS model.



(B) Solution of the SIS model with vital dynamics.

Fig. 2: SIS model.

This assumption is generally mentioned as the mass-action principle. Mathematically, the SI model (Fig. 1A) can be expressed as a system of differential equations:

$$dS/dt = -\beta SI/N, \tag{1}$$
$$dI/dt = \beta SI/N, \tag{2}$$

with initial conditions:

$$S(0) = S_0 \geq 0, \quad I(0) = I_0 \geq 0, \tag{3}$$
$$S_0 + I_0 = N_0 = N = S(t) + I(t). \tag{4}$$

Here, initial population $N_0 = N$ as the population as a whole is supposed to remain constant that means there are no births, deaths, or migration in or out of the population. This model helps to clarify the fundamental dynamics of infectious diseases and can be used to make predictions about the potential spread of an outbreak. However, it neglects many important influencing factors like the infectious period, incubation period, and the effect of interventions such as vaccination and quarantine.

Solutions of the model Eqs. (1) and (2) are obtained by using $S + I = N$ in Eq. (2), separating variables to integrate and applying initial conditions given as:

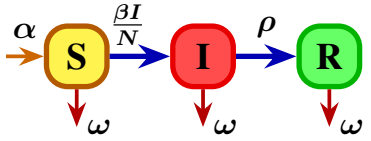
$$I(t) = \frac{I_0 N}{I_0 + S_0 e^{-\beta t}}, \tag{5}$$
$$S(t) = N - I(t). \tag{6}$$

The graphical solution (Fig. 1B) of SI model (for hypothetical data and parameters: $N_0 = 100000$, $I_0 = 1000$, $\beta = 0.3$) shows that the number of susceptible individuals declines exponentially over time, and the number of infectious individuals rises exponentially until the entire population becomes infected.

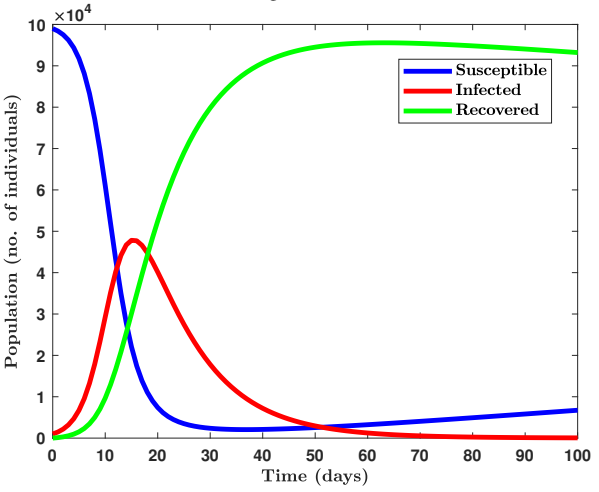
The disease free equilibrium (DFE) in SI model is simply when whole population are susceptible and there are no infectious individuals. Mathematically, when $S = S_0$ and $I = 0$, so that DEF is $(S_0, 0)$. For this model, $\mathcal{R}_0 = \beta N$ because each infectious individual can directly infect βN susceptible individuals and there are no recovery or removal processes.

B. Susceptible-Infected-Susceptible (SIS) model

SIS epidemic model assumes that the individuals in infected compartment reappear to the susceptible compartment. It is appropriate for the diseases like gonorrhea or chlamydia (sexually transmitted diseases)

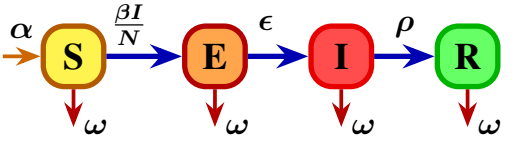


(A) Flow diagram of SIR model

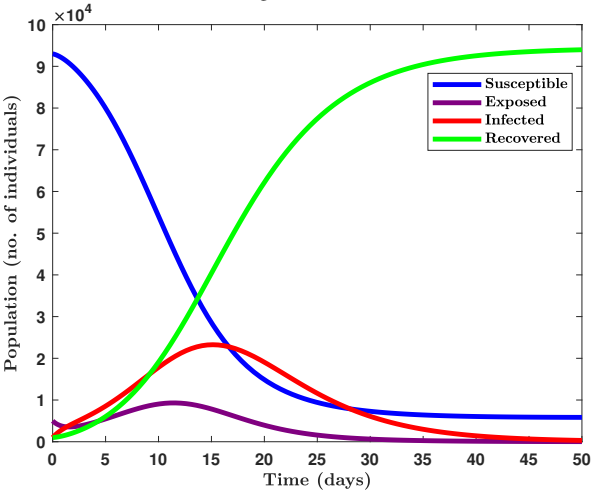


(B) Solution of SIR model with vital dynamics

Fig. 3: SIR model.



(A) Flow diagram of SEIR model.



(B) Solution of SEIR model with vital dynamics.

Fig. 4: SEIR model.

and common cold as they normally have repetitive infections.

The mathematical expression of SIS epidemic model (Fig. 2A) with birth, natural death and recovery rate into a closed population ($\alpha = \omega$) can be formulated as follows [54]:

$$dS/dt = \alpha N - \beta SI/N - \omega S + \nu I, \tag{7}$$
$$dI/dt = \beta SI/N - (\nu + \omega)I, \tag{8}$$

with initial conditions:

$$S(0) = S_0 \geq 0, \quad I(0) = I_0 \geq 0, \tag{9}$$
$$S_0 + I_0 = N_0 = N = S(t) + I(t). \tag{10}$$

One can obtain the SIS model without vital dynamics by just ignoring the birth rate and death rate term.

After normalization by substituting $s = S/N$, $i = I/N$, the equations (7) to (10) become:

$$ds/dt = \alpha - \beta si - \omega s + \nu i, \tag{11}$$
$$di/dt = \beta si - (\nu + \omega)i, \tag{12}$$
$$s(0) = s_0 \geq 0, \quad i(0) = i_0 \geq 0, \tag{13}$$
$$s_0 + i_0 = 1 = s(t) + i(t). \tag{14}$$

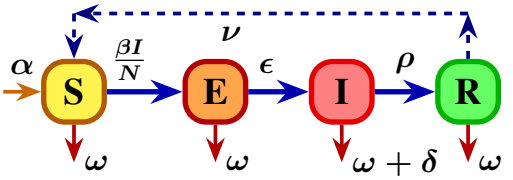
Using $s(t) = 1 - i(t)$ into Eq. (12) and solving the resulting Bernoulli's equation, we get the analytic solution of the model without vital dynamics ($\alpha = 0$, $\omega = 0$) as follows:

$$i(t) = \frac{i_0 \xi}{\xi e^{-\xi t + \beta i_0 (1 - e^{-\xi t})}}, \tag{15}$$
$$s(t) = 1 - i(t), \tag{16}$$

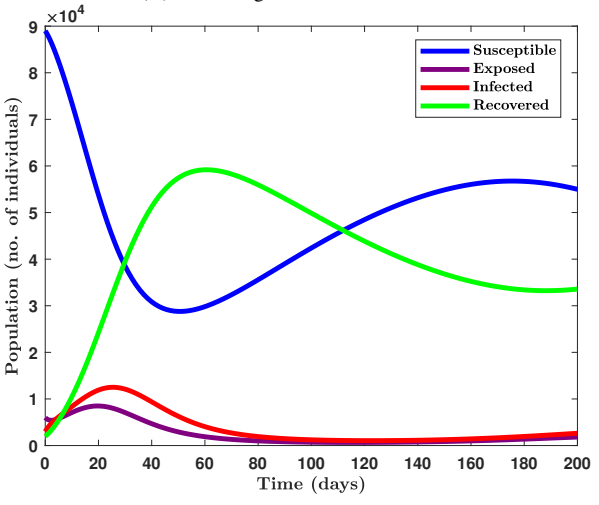
where $\xi = \beta - \nu$. For the case of the model with vital dynamics numerical approaches like Euler's and Runge-Kutta method are more appropriate. For the model with vital dynamics $\mathcal{R}_0 = \frac{\beta}{\nu + \omega}$, whereas considering $\omega = 0$ gives the value of $\mathcal{R}_0$ for the model without vital dynamics. The graphical solution (Fig. 2B) of SIS model (for hypothetical data and parameters: $\beta = 0.8$, $\nu = 0.2$, $\alpha = \omega = 0.02$, $N_0 = 100000$, $I_0 = 1000$) indicates that as the disease spreads, the number of infected people increases and susceptible people decreases. Eventually, the number of infected individuals begins to decline and number of susceptible begins to increase slowly as the individuals recover from the disease and become susceptible again.

C. Susceptible-Infected-Removed (SIR) model

The SIR model assumes that once an individual recovers from the infection, it suffers from immunity to the disease and remains so for the rest of its life.



(A) Flow diagram of SEIRS model.



(B) Solution of SEIRS model with vital dynamics.

Fig. 5: SEIRS model.

Normally the recovered individuals are not returned in the susceptible population. This model is normally suitable for the diseases having lifelong immunity after recovery such as mumps, measles, rubella and pertussis.

The SIR model with vital dynamics (Fig. 3A) can be expressed as a system of differential equations as follows [54]:

$$dS/dt = \alpha N - \beta SI/N - \omega S, \tag{17}$$
$$dI/dt = \beta SI/N - (\rho + \omega)I, \tag{18}$$
$$dR/dt = \rho I - \omega R, \tag{19}$$

with initial conditions:

$$\begin{aligned} &S(0) = S_0 \geq 0, \quad I(0) = I_0 \geq 0, \\ &R(0) = R_0 \geq 0, \\ &S_0 + I_0 + R_0 = N_0 = N. \end{aligned} \tag{20}$$

For the model without vital dynamics $\alpha = \omega = 0$ and that with vital dynamics assuming $\alpha = \omega$ in non-varying population, $S(t) + I(t) + R(t) = N$ (constant) gives $dS/dt + dI/dt + dR/dt = 0$.

Normalizing the model equations (17) to (19) by substituting $s = S/N$, $i = I/N$, $r = R/N$, the above model becomes:

$$ds/dt = \alpha - \beta si - \omega s, \tag{21}$$
$$di/dt = \beta si - (\rho + \omega)i, \tag{22}$$
$$dr/dt = \rho i - \omega r, \tag{23}$$
$$s(0) = s_0 \geq 0, \; i(0) = i_0 \geq 0, \; r(0) = r_0 \geq 0, \tag{24}$$
$$s_0 + i_0 + r_0 = 1. \tag{25}$$

Analytic solutions of the equations (21)–(23) without vital dynamics ($\alpha = \omega = 0$) are given by [55]:

$$s(t) = s_0 e^{-\xi(t)}, \tag{26}$$
$$r(t) = r_0 + \rho\xi(t)/\beta, \tag{27}$$
$$i(t) = 1 - s(t) - r(t), \tag{28}$$

where $\xi(t) = \beta \int_0^t i(\hat{t})d\hat{t}$, $\xi(0) = 0$.

For the model without vital dynamics, the threshold quantity $\mathcal{R}_0$ is given by, $\mathcal{R}_0 = \frac{\beta s_0}{\rho}$ [56]. Re-scaling s by S/N, $\mathcal{R}_0 = \frac{\beta S_0}{N\rho} = \frac{\beta}{\rho}$, since $S_0 \approxeq N$ in entirely susceptible population (N). Whereas the basic reproduction number for SIR model with vital dynamics is given by $\mathcal{R}_0 = \frac{\beta}{\rho+\omega}$ [57]. It is more convenient to get numerical solutions than analytic solutions of SIR model with vital dynamics because of the non-linearity and complex structure [58]. Generally numerical solutions are obtained by using Euler's, modified Euler's, finite difference and RK-4 methods etc. One common numerical approach to solve such type of system of ordinary differential equations of first-order is Euler's method. The Euler's method works by approximating the solution at each time step with size $\Delta t = \tau$ using the values of the variables prior to the previous time step. The Euler's approximation formulas for system Eqs. (21)–(23) are:

$$s(t + \tau) = s(t) - \tau[\alpha - \beta s(t)i(t) - \omega s(t)], \tag{29}$$
$$i(t + \tau) = i(t) + \tau[\beta s(t)i(t) - (\rho + \omega)i(t)], \tag{30}$$
$$r(t + \tau) = r(t) + \tau[\rho i(t) - \omega r(t)]. \tag{31}$$

The simulation is started by setting the initial values S_0, I_0, R_0 and estimated parameters $\alpha, \beta, \omega, \rho$. Then, the Euler's method is used to update normalized state variables s, i and r at each time step until the desired time horizon is reached. The graphical solution (Fig. 3B) of the SIR model with vital dynamics (for hypothetical data and parameters: $\beta = 0.5$, $\rho = 0.1$, $\alpha = \omega = 0.01$, $N_0 = 100000$, $I_0 = 1000$, $R_0 = 0$) shows that susceptible individuals fall off over time, whereas the number of infectious people rises at first and then falls as people recover and become immune. The number of recovered individuals increases over time until the entire population becomes immune.

D. Susceptible-Exposed-Infected-Removed (SEIR) model

The SEIR epidemic model is considered an advancement of SIR model and is generally used to study infectious disease transmission with a latent period during which an infected individual is not yet contagious. The suspension between acquiring an infection and becoming infectious can be characterized by inserting a latent/exposed compartment, E. The diseases with a considerable incubation period such as chicken pox, dengue, COVID-19 fall into this category.

Based on the flow diagram (Fig. 4A) of the SEIR model, the dynamics of the compartments can be described by the following system of differential equations [59]:

$$dS/dt = \alpha N - \beta SI/N - \omega S, \tag{32}$$
$$dE/dt = \beta SI/N - \epsilon E - \omega E, \tag{33}$$
$$dI/dt = \epsilon E - \rho I - \omega I, \tag{34}$$
$$dR/dt = \rho I - \omega R, \tag{35}$$

with

$$S(0) = S_0 \geq 0, \quad E(0) = E_0 \geq 0,$$
$$I(0) = I_0 \geq 0, \quad R(0) = R_0 \geq 0, \tag{36}$$
$$S(t) + E(t) + I(t) + R(t) = N(t),$$
$$S_0 + E_0 + I_0 + R_0 = N_0. \tag{37}$$

If the terms containing α and ω are neglected from these equations, then the resulting system performs as the SEIR model without vital dynamics well.

Instead of dealing with the system of ODEs (32) to (35) with known population $N = N_0 e^{(\alpha-\omega)t}$, the transformations $s = S/N = y_1$, $e = E/N = y_2$, $i = I/N = y_3$, $r = R/N = y_4$ (that represent the fractions of S, E, I, R) are applied and the transformed system is formulated as:

$$ds/dt = dy_1/dt = \alpha - \beta si - \alpha s, \tag{38}$$
$$de/dt = dy_2/dt = \beta si - \epsilon e - \alpha e, \tag{39}$$
$$di/dt = dy_3/dt = \epsilon e - \rho i - \alpha i, \tag{40}$$
$$dr/dt = dy_4/dt = \rho i - \alpha r, \tag{41}$$
$$y_j(0) = (y_j)_0 \geq 0, \quad j = 1, 2, 3, 4, \tag{42}$$
$$\sum_{j=1}^{4} y_j(t) = 1. \tag{43}$$

The value of $\mathcal{R}_0$ for the SEIR model with vital dynamics can be obtained by deriving the next generation matrix for infection classes (E and I) introduced by Diekmann et al. [60]. Derivation by this approach gives $\mathcal{R}_0 = \frac{\beta\epsilon}{(\epsilon+\omega)(\rho+\omega)}$ which is justified with the calculation for $\mathcal{R}_0$ in [57, 61]. For the model without vital dynamics, $\mathcal{R}_0$ can be found by the similar manner as stated in SIR model.

As the model includes system of nonlinear differential equations that have no convenient method for exact analytic solution, numerical solutions are appropriate. One of the most common methods to solve the SEIR model Eqs. (38)–(41) is the RK-4 method, which accepts the derivatives term as functions of t, $s = y_1$, $e = y_2$, $i = y_3$, and $r = y_4$, so that $f_{y_j}(t, y_1, y_2, ..., y_4) = \frac{dy_j}{dt}$, $j = 1, 2, 3, 4$.

RK-4 formulas to update the approximate solutions for y_1, y_2, y_3, y_4 at each iteration $n = 0, 1, 2, 3, 4, \ldots$ with time step size $\Delta t = t_n - t_{n-1} = \tau$ are given by:

$$(y_j)_{n+1} = (y_j)_n + \frac{\tau}{6}\big((k_j)_1 + 2(k_j)_2 + 2(k_j)_3 + (k_j)_4\big), \tag{44}$$

where $j = 1, 2, 3, 4$ and

$$(k_j)_1 = f_{y_j}\big(t_n, (y_1)_n, (y_2)_n, (y_3)_n, (y_4)_n\big), \tag{45}$$
$$(k_j)_2 = f_{y_j}\Big(t_n + \frac{\tau}{2}, (y_1)_n + \frac{(k_1)_1}{2}, (y_2)_n + \frac{(k_2)_1}{2}, \ldots, (y_4)_n + \frac{(k_4)_1}{2}\Big), \tag{46}$$
$$(k_j)_3 = f_{y_j}\Big(t_n + \frac{\tau}{2}, (y_1)_n + \frac{(k_1)_2}{2}, (y_2)_n + \frac{(k_2)_2}{2}, \ldots, (y_4)_n + \frac{(k_4)_2}{2}\Big), \tag{47}$$
$$(k_j)_4 = f_{y_j}\Big(t_n + \tau, (y_1)_n + (k_1)_3, (y_2)_n + (k_2)_3, \ldots, (y_4)_n + (k_4)_3\Big). \tag{48}$$

Eq. (44) gives the values of $y_1, y_2, ..., y_4$ that means the number of individuals in different compartments at time $t_1, t_2, ..., t_n$.

The graphical solution (Fig. 4B) of the SEIR model with vital dynamics (for hypothetical data and parameters: $\beta = 0.6$, $\rho = 0.2$, $\epsilon = 0.6$, $\alpha = 0.00005$, $\omega = 0.00002$, $N_0 = 100000$, $I_0 = 1000$, $E_0 = 5000$, $R_0 = 1000$) demonstrates that susceptible individuals decreases as they remove into subsequent compartments one after the other over time, while the exposed individuals rise in number at first and then fall off as they become infectious. Similar to the exposed individuals, the number of infectious persons first go up and then down as they recover with permanent immunity. The number of recovered individuals increases over time until the whole population becomes immune.

E. Susceptible-Exposed-Infected-Removed-Susceptible (SEIRS) model

The SEIRS epidemic model, a slight modification of the SEIR model includes a temporary loss of immunity after recovery of the infected individuals who might revert to susceptibility after a certain period of time. Examples of illnesses with protracted incubation periods and for which infection only results in transient immunity are rotavirus and malaria. Model equations for SEIRS model are as given:

$$dS/dt = \alpha N - \beta SI/N + \nu R - \omega S, \quad (49)$$

$$dE/dt = \beta SI/N - \epsilon E - \omega E, \quad (50)$$

$$dI/dt = \epsilon E - \rho I - (\omega + \delta)I, \quad (51)$$

$$dR/dt = \rho I - \nu R - \omega R, \quad (52)$$

with

$$S(0) = S_0 \geq 0, \quad E(0) = E_0 \geq 0,$$
$$I(0) = I_0 \geq 0, \quad R(0) = R_0 \geq 0, \quad (53)$$
$$S(t) + E(t) + I(t) + R(t) = N(t),$$
$$S_0 + E_0 + I_0 + R_0 = N_0. \quad (54)$$

Flow diagram and solution of the model are presented in (Fig. 5A) and (Fig. 5B) respectively.

$\mathcal{R}_0$ is derived in the similar method as in SEIR model, but as the diseases-related fertility rate δ is also included in this model, $\mathcal{R}_0 = \frac{\beta\epsilon}{(\epsilon+\omega)(\rho+\omega+\delta)}$ [62].

The solution for the SEIRS model can also be obtained similarly as in SEIR model using the Runge-Kutta method of fourth order. Minor differences occur in RK-4 formula because of the inclusion of immunity loss rate and infection-induced death rate in the later case. The graphical solution (Fig. 5B) of the SEIRS model (for hypothetical data and parameters: $\beta = 0.33$, $\rho = 0.15$, $\nu = 0.01$, $\alpha = 5 \times 10^{-5}$, $\omega = 2 \times 10^{-5}$, $\delta = 0.01$, $\epsilon = 0.25$, $N_0 = 100000$, $I_0 = 3000$, $E_0 = 6000$, $R_0 = 2000$) shows a different scenario than the SEIR model. In this case, the number of susceptible individuals decreases for a certain time and then begins to increase because recovered individuals with short-term immunity become susceptible again after a certain period. This causes a decrease in recovered individuals after that time. The exposed and infected individuals first increase as the susceptible individuals decrease and then begin to decrease slightly.

F. Other modifications on SEIR model

Epidemiologists have made valuable efforts on modifications to SEIR epidemic model to create more accurate models for the dynamics of different diseases.

Parameters	Meanings
$f_i = f_i(t)$	Meteorological factors ($i = 1, 2, 3, \ldots$ as required with their corresponding units)
β	Birth rate (day^{-1})
δ	Natural death rate (day^{-1})
$\phi = \phi(t, f_i)$	Transmission rate from S to E (day^{-1})
$\psi = \psi(t, f_i)$	Infection rate from E to I_s (day^{-1})
$\rho = \rho(t, f_i)$	Infection rate from E to I_a (day^{-1})
$\mu = \mu(t, f_i)$	Rate from E to Q (day^{-1})
$\alpha = \alpha(t, f_i)$	Infection rate from Q to I_s (day^{-1})
$\sigma = \sigma(t, f_i)$	Infection rate from Q to I_a (day^{-1})
r	Recovery rate from Q (day^{-1})
κ	Rate of I_s being hospitalized (day^{-1})
h	Rate of I_s being home isolated (day^{-1})
p	Recovery rate from I_a (day^{-1})
m	Rate of I_a being home isolated (day^{-1})
l	Rate of I_a being hospitalized (day^{-1})
$\eta = \eta(t, f_i)$	Death rate from H_m (day^{-1})
$\nu = \nu(t, f_i)$	Rate of H_m being hospitalized (day^{-1})
$q = q(t, f_i)$	Recovery rate from H_m (day^{-1})
$\epsilon = \epsilon(t)$	Death rate from H (day^{-1})
$n = n(t)$	Recovery rate from H (day^{-1})

Table 3: Model parameters for CA-SEIR Model.

The major modifications are constructed by including additional compartments with additional parameters. One modification to the SEIR model is to include age structured compartment [63], which is done by distributing the population into different age clusters and assigning different infection and recovery rates to each group. The SEIR model can be modified to address the effects of virus variants [64]. This can be done by adjusting the infection and recovery rates based on the variant's characteristics, such as its transmission rate and severity (represented by case fatality rate, hospitalization rate, nature and intensity of symptoms). Another modification to the SEIR model is to include the vaccinated compartment [64, 65] of the population who have been vaccinated and are therefore less likely to get infected or transmit the disease. There are many other modifications or extensions [64, 66–69] including the compartments for Quarantined (Q) or Home isolated (H_m), Hospitalized (H), Dead (D), Protective (P), Asymptomatic and Symptomatic Infected (I_a, I_s), Laboratory tested and untested (L_t, L_u) etc. As different meteorological conditions significantly impact the spread of infectious diseases, implementation of these factors on different models discussed above can provide a valuable tool for studying their effects on the diseases.

Compartmental Augmentation of SEIR Model (CA-SEIR Model): As a compartmental augmentation of the SEIR (CA-SEIR) model, we propose a modi-

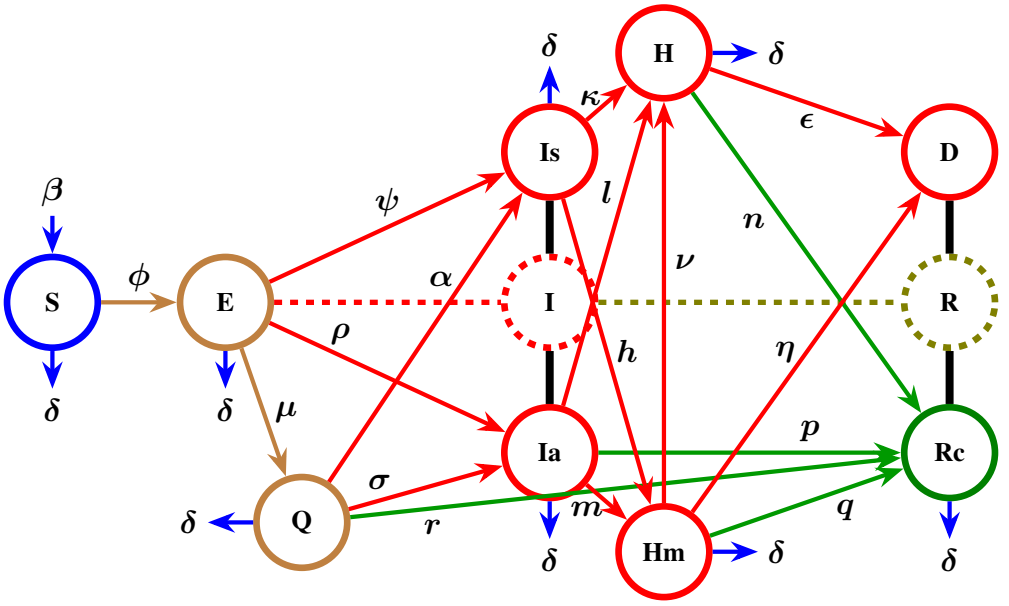


Fig. 6: Diseases transmission network for CA-SEIR model.

fication of SEIR model for airborne diseases such as COVID-19 by extending the compartments (Fig. 6) and introducing the parameters as given in Table 3.

Expressing the infectious diseases transmission network (Fig. 6) mathematically, the governing differential equations are:

$$\begin{aligned}
\frac{dS}{dt} &= \beta N - \delta S - \phi\frac{S(I_s+I_a)}{N},\\
\frac{dE}{dt} &= \phi\frac{S(I_s+I_a)}{N} - (\delta+\psi+\rho+\mu)E,\\
\frac{dQ}{dt} &= \mu E - (\alpha+\sigma+r+\delta)Q,\\
\frac{dIs}{dt} &= \psi E + \alpha Q - (\kappa+h+\delta)I_s,\\
\frac{dIa}{dt} &= \rho E + \sigma Q - (m+l+p+\delta)I_a,\\
\frac{dH}{dt} &= \kappa I_s + lI_a + \nu H_m - (\epsilon+n+\delta)H,\\
\frac{dHm}{dt} &= mI_a + hI_s - (q+\delta+\eta+\nu)H_m,\\
\frac{dRc}{dt} &= nH + pI_a + qH_m + rQ - \delta R_c,\\
\frac{dD}{dt} &= \epsilon H + \eta H_m,
\end{aligned}$$

with the conditions that at any time t and at $t = 0$ (initially), sum of individuals in all compartments $= N$ and N_0 respectively.

The model consists of three types of parameters: constant, time-dependent (function of t) and time-meteorological-dependent (function of t and f_i). Other factors such as interventions, vaccinations, hospital facilities, which may influence the parameters, are not considered, as the model focuses solely on meteorological factors.

The time dependent parameters may be associated with meteorological factors, weather factors, control measure factors, vaccination rate, hospital facilities, and other health condition of the patients. Estimating parameters can be crucially complicated when trying to include all factors in such models. So, focusing on only some meteorological factors will be preferable. Once the parameters are estimated, the solution techniques will be a numerical method such as Euler's or Runge-Kutta methods. As this is a review paper, it does not include a detailed theoretical and numerical analysis of the proposed model. A comprehensive study will be undertaken in future research.

V. Research Gaps and Discussion

Major gaps in the research on compartmental epidemic models with meteorological factors are limited consideration of real time data, air properties, seasonal variations and extreme weather events, multi-scale atmospheric modeling, validation of meteorologically-modified models, variability in human population and behaviors, estimation of epidemic parameters, multiple nonlinear diseases dynamics, and uncertainties in meteorological analysis.

There are several studies (some are discussed in the background section) on deterministic compartmental models to investigate the meteorological effects on the transmission dynamics of diseases. Most models assume homogeneity within each compartment, disregarding individual characteristics (age, sex, behavior, actions, etc.) that can affect disease transmission and progression [70]. Some use constant transition rates with exponentially distributed sojourn times [71], but interventions like immunization, social separation, and quarantine can impact the rate at which diseases spread. Zhou et al. [72] reported a negative association between temperature and humidity with influenza, influencing virus shedding, survival, transport, and host immunity. But the limited data length and surveillance gaps in certain countries hinder the ability to test the model's universality and long-term stability. Chang et al. [73] concluded that temperature and precipitation can change the dynamics of vectors, but they had some gaps, such as not including different serotypes transmission, investigating with a constant temperature, and not taking spatial effects. Yang et al. [14] examined the effect of weather on measles incidence and observed nonlinear effects of temperature and relative humidity, but they had some limitations such as, difficulty in generalizing the study, overestimated the incidence, and unavailability of the wind speed and precipitation data. Lowen and Steel [7] indicated that absolute humidity and temperature affect influenza seasonality, without sufficiently clarify the potential mechanisms that alter transmission. Ai et al. [74] found nonlinear and relatively weak effects of meteorological factors on COVID-19, mainly with lower temperature and RH increasing the risk of infection. Nevertheless, using country-level averages for meteorological data in large countries may dilute accuracy, highlighting the need for studies at the local level for more precise results. Liu et al. [75] used Spearman correlation analysis to examine the relationships between Hand-Foot-Mouth-Disease (HFMD) and meteorological factors, finding a positive association with precipitation, temperature and humidity, but a negative association with wind speed. However, the study's use of annual average rates for HFMD and corresponding annual meteorological averages did not account for lag effects. Additionally, the Generalized Additive Model (GAM) employed in the study had limitations, including complex parameter settings and sensitivity of results to parameter selection.

Validating models that include meteorological factors is complex due to the interplay between disease dynamics and atmospheric conditions which causes continuing challenges on developing valid models combining both epidemiological and meteorological data [13,76]. Lindsay and Birley [77] suggested to authorize the accuracy of the predicting models and to approve the assumptions. While meteorological factors have received significant attention in epidemic modeling, other variables such as pollution, global warming, rain, climatic change and UV radiation can also affect disease transmission. Wu et al. [51] considered only temperature and relative humidity as climate factors, not covering the effect of intervention policies on the outbreaks and compromising for real-time data of COVID-19 cases and some ecological fallacies. Li et al. [53] introduced a machine learning technique on an improved SEIR model of COVID-19 to investigate the relationship between environmental influences and the spread of viruses, but they lacked of considering comprehensiveness of environmental and additional elements including viral variants, biodiversity, and air pollution, and population immunity. Estimating the epidemic parameters by fitting available data becomes more challenging with the attachment of meteorological factors. Naserpor et al [44] highlighted the impact of temperature, precipitation, humidity, and other environmental conditions using modified SIR model on the influenza transmission. However, the use of average climate data and new case data from the vast country limited the model's performance and the accuracy of the estimation of parameter. Haga et al [6] concluded that increased humidity and temperature lower the relative risk of COVID-19. But their statistical study did not cover a long study period and government policies.

Though certain researchers [7,8,78–81] have tried to address the gaps mentioned above, it still seems more appropriate to conduct thorough and updated research on some gaps that can be filled based on the research problem of the model, the quality of data, and their availability. To access up-to-date real-time meteorological data, implementation of data integration technique and update of data source mechanism can be applied. Oversimplification of meteorological effects can be handled by incorporating non linear functions or machine learning algorithms to model the complex dependencies between meteorological factors and pathogens survival rate. Air pollution data can be included in the model by applying their spatial variations. Difficulties in estimating or optimizing epidemic parameters can be addressed by applying mathematical, statistical, or hybrid methods such as the Bayesian method, machine learning algorithms, genetic algorithms, and particle swarm optimization.

VI. Applications

Epidemic models with meteorological factors have a wide-range of applications as they offer important ideas into how infectious diseases spread concerning atmospheric conditions and what to do for prevention. They can predict and monitor disease outbreaks on current and forecasted meteorological conditions, helping to manage public health plans and allocate resources for disease investigation, prevention, and control. For diseases transmitted by vectors and air, the models help to determine the correlation of diseases spread with air temperature, wind pattern, humidity, air circulation, etc. Understanding vector movement and pathogens' survival or dispersal can help predict the risk of such diseases in different environments. The models provide geographical spread analysis and guide targeted intervention and prevention approaches, and can assess how changes in climatic conditions and seasonal variation might alter disease transmission patterns, serving to adapt public health strategies to changing environments. The models are useful for informing public health policy and directing resource allocation decisions by assessing the efficacy of various intervention techniques, including social distance, vaccination, lock down, and quarantine. Travel advisories and limits can be informed using epidemic models to evaluate the risk of disease transmission across different locations, considering various weather and climate conditions. Epidemic models can be used to design and simulate clinical trials for new treatments or vaccines. They help estimate an outbreak's economic impact, including the costs associated with healthcare, lost productivity, and supply chain disruptions. The formulation of research hypotheses can be directed through these models by identifying possible links between disease dynamics and meteorological factors, leading to further studies and data collection. Encouraging cross-disciplinary collaboration among epidemiologists, meteorologists, mathematicians, and other experts; these models can lead to innovative solutions for tackling infectious diseases.

VII. Conclusions

It seems that many researches on deterministic compartmental model do not fully cover in incorporating more realistic meteorological factors, human behaviors, model validation, parameters estimation, real-time prediction of the diseases spread, and spatial heterogeneity. By addressing the research gaps, new researches can improve the understanding of diseases spread through environment and develop more effective strategies for controlling their transmission. This can be done by conducting empirical studies to understand the impact of various meteorological and climate factors along with individual-level-characters. Taking social norms and national intervention policies, developing spatially explicit models with environmental and behavioral factors, incorporating individual-level factors, and formulating real time models can predict the real-time transmission of infectious illnesses.

The proposed (CA-SEIR) model which is modification of deterministic SEIR model has nine compartments and a large number of parameters. The main research problems corresponding to this model are to categorize and determine the epidemic parameters with the help of epidemic and meteorological data, to find the numerical solutions with stability analysis, to derive basic reproduction number, and to suggest the control measures. The complexity of the model can be dealt with various data-driven, mechanistic or hybrid approaches including Sequential Monte Carlo, Bayesian Inference with Markov Chain Monte Carlo, Recurrent Neural Networks with Long Short-Term Memory, Generalized Additive Models, Nonlinear Least Squares, Extended Kalman Filter, along with some advanced optimization techniques like Particle Swarm Optimization and Genetic Algorithm. Finding the particular settings and contributing variables to an epidemic or pandemic as well as creating effective control measures are among the research's goals. Future research will concentrate on verifying these assumptions by mathematical theories, laboratory tests, or field investigations because many models rely on assumptions that might not adequately represent real-world situations.

Acknowledgements

Russell Alpizar-Jara is supported by Research Center for Mathematics and Applications (CIMA), through the project UIDB/04674/2020 of FCT – Fundação para a Ciência e a Tecnologia, Portugal.

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