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Developing an SEIHR epidemiological model featuring saturated incidence rate and investigating its stability

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Abstract

Each and every person will inevitably come into contact with infectious diseases over their lifetime. This unavoidable phenomena is a characteristic of all people. Ideally, the effects of such diseases are still controllable, but under unfavourable circumstances, they may worsen and become full-fledged outbreaks. As a result, this article tries to offer an SEIHR model whose main goal is to reduce and stop the transmission of infectious diseases. Notably, infectious diseases continue to plague many countries, represented by the global threat of Covid-19, underlining the urgent need for efficient control methods. This model stands out because it carefully takes into account the saturated incidence rate of infectivity. The concept also divides the infected population into two different groups, enabling more focused and customized medical interventions. The article begins by closely examining the positivity and boundedness of the model, then conducts a thorough analysis of the fundamental reproductive number. The model's stability is then categorically proven at both equilibrium points. In summary, the article concludes with convincing numerical simulations that validate the suggested model, providing convincing evidence of its effectiveness as a whole. In conclusion, the suggested SEIHR model, which is supported by a wealth of empirical evidence confirming its efficacy and optimality, represents a significant advancement in the management and containment of infectious diseases. It offers a promising route for addressing the enduring threat of infectious diseases on a global scale, and its unique form and extensive analysis highlight its potential to alter our approach to infectious disease control.

Keywords: Infectious diseases; SEIHR model; Saturated Incidence rate; Outbreak Prevention; Efficacy validation

1. Introduction

Mathematical modeling stands as a fundamental pillar in epidemiology, furnishing a sturdy method for comprehending and foretelling outbreaks. Within infectious disease exploration, these models furnish an organized platform for scrutinizing the intricate dynamics of transmission, encompassing a myriad of influencing factors. At the core of these models lie incidence rates, gauging the velocity of new case emergence within a populace. These rates serve as linchpins in seizing the temporal nuances of infectious ailments, shaped by populace magnitude, environmental variables, and public health interventions. Through the integration of saturated incidence rates, models recognize that transmission eventually reaches equilibrium owing to factors such as immunity or a dwindling pool of susceptible individuals.

Beyond comprehending disease dynamics, mathematical modeling plays a crucial role in shaping public health policies and interventions. Through simulating various scenarios and forecasting potential outcomes, it facilitates decision-making, allocation of resources, and formulation of strategies, encompassing control measures, while also evaluating their efficacy.

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In essence, the synergy between mathematical models and incidence rates, coupled with their nuanced integration, particularly through saturated incidence rates, proves indispensable in unraveling the intricacies of infectious disease dynamics. Within the realm of infectious diseases, this approach not only enriches theoretical comprehension but also empowers practical implementations, enabling a more informed and targeted response to mitigate their impact on global health.

In 2020, a study published in Chaos, Solitons & Fractals (Ndaïrou, 2020) employed mathematical modeling to delve into the transmission dynamics of COVID-19, focusing specifically on Wuhan. By integrating a multitude of characteristics and dynamics into their model, the authors likely conducted a comprehensive analysis of the disease's transmission.

Furthermore, Mammeri Y's research, featured in Computational and Mathematical Biophysics (Mammeri, 2020), presents a reaction-diffusion system aimed at aiding readers in understanding the impacts of implementing measures against the COVID-19 pandemic. This study probably utilizes a diffusion-based SEIR-type model to assess the geographical spread of the virus in France.

The study by Ouedraogo et al. (2021) explores the stability analysis of an SEIHR model concerning infectious disease transmission. Additionally, the authors scrutinize the stability and equilibrium behavior of the proposed model. This piece was published in Non-autonomous Dynamical Systems (2021).

A pre-print titled "Investigating the Impact of Asymptomatic Carriers on COVID-19 Transmission" by Aguilar JB et al. (2020) is available on medRxiv. This preprint delves into the influence of asymptomatic carriers on the kinetics of COVID-19 transmission, potentially shedding light on the role of individuals without visible symptoms in spreading the virus.

In the work by Niu et al. (2021), published in Nonlinear Dynamics, a stochastic SEIHR model is introduced to accommodate the inherent variability in COVID-19 data. The authors employ a probabilistic modeling approach to address the pandemic's fluctuating characteristics.

According to research conducted by Malagutti RM et al., titled "SEIHR Epidemic Model Applied to the COVID-19 Outbreak in Sao Paulo" (Malagutti, R. M., & Eisencraft, M., 2021), utilizing the SEIHR model can aid in evaluating the dynamics of the COVID-19 outbreak. The findings published could provide valuable insights into the progression of the disease within a specific region.

In a study titled "Determining Important Parameters in the Spread of Malaria through Sensitivity Analysis of a Mathematical Model" (Chitnis et al., 2008), published in the Bulletin of Mathematical Biology in 2008 by Chitnis N et al., although not directly focused on COVID-19, a comparative perspective might be gleaned. Through a sensitivity analysis, the authors aimed to identify pivotal factors influencing the spread of malaria.

The article "Optimal Control of SEIHR Mathematical Model of COVID-19" (Yoda et al., 2023) by Yoda Y et al. delves into determining the most effective control measures for the COVID-19 SEIHR model. This study proposes methods to enhance strategies and mitigate the impacts of the disease.

For the examination of local stability in epidemic models, Adom-Konadu A et al. (Adom-Konadu et al., 2022) employed the corollary of Gershgorin’s Circle Theorem. Their work may elucidate the stability characteristics of epidemiological models.

In 2022, Kim YR et al. published an article titled "A Model of COVID-19 Pandemic with Vaccines and Mutant Viruses" in PLOS ONE (Kim et al., 2022). In the context of the COVID-19 pandemic, their paper proposes a model that incorporates vaccinations and mutant viruses, exploring how disease dynamics are influenced by vaccination strategies.

Human migration is integrated into an SEIHR model in the research by Niu R et al. (Niu et al., 2020), published in IEEE (2020), aiming to enhance understanding of the spatial dynamics of the COVID-19 pandemic.

The study by Rodrigues HS et al., "Sensitivity Analysis in a Dengue Epidemiological Model" (Rodrigues et al., 2013), published in Conference Papers in Mathematics (2013), provides insights into sensitivity analysis concerning a dengue epidemiological model.

In summary, the comprehensive literature review conducted offers a nuanced examination of numerous mathematical models utilized in epidemiology, with a particular emphasis on the significant implications of infectious diseases. These

studies collectively underscore the pivotal role of mathematical modeling in shaping public health strategies, serving as the impetus for this article to elucidate the dynamic complexities of infectious diseases that necessitate accurate comprehension and effective management.

Consequently, this article diverges from conventional SEIHR (Susceptible-Exposed-Infectious-Hospitalized-Recovered) models by placing a distinct emphasis on the incidence rate. Unlike many models that predominantly track individuals' progression through different disease states, this approach prioritizes the incidence rate to project future at-risk individuals. This shift highlights the preventive aspect of the research, aiming to identify and anticipate potential infections among populations.

Through vigilant monitoring of the incidence rate, the study aims to issue early warnings about potential outbreaks and pinpoint populations at elevated risk. This proactive stance is vital for comprehending the evolving disease dynamics and enables stakeholders to enact timely and targeted measures. Rather than merely reacting to existing cases, focusing on the incidence rate provides a forward-looking perspective, guiding preventive actions and interventions.

Moreover, by integrating the incidence rate into the modeling framework, the study endeavours to deepen our understanding of disease transmission dynamics. This enhanced insight into the underlying mechanisms of spread can facilitate the formulation of more efficacious strategies for disease control and mitigation. By pre-emptively identifying areas or populations with high incidence rates, resources and interventions can be strategically allocated, maximizing their effectiveness in curtailing the disease's spread.

In essence, the emphasis on the incidence rate in this study embodies a proactive and precautionary approach to disease modeling. By predicting future at-risk individuals and comprehending the dynamics of disease transmission, it aims to equip decision-makers with the requisite knowledge to implement timely and targeted interventions, thereby contributing to enhanced disease control and management.

2. Materials and Methods

2.1. Formulation of the Model

The proposed SEIHR epidemiological model is given by

$$\begin{aligned} S' &= \Lambda - \frac{\beta_1 SI}{1+\alpha_1 I} - \beta_2 SE - \mu S \\ E' &= \beta_2 SE - (\gamma + \lambda + \mu) \\ I' &= \frac{\beta_1 SI}{1+\alpha_1 I} + \gamma E - (\eta + \delta_1 + k_1 + \mu) I \qquad \text{.....(1)} \\ H' &= \eta I - (\delta_2 + k_2 + \mu) H \\ R' &= \lambda E + \delta_1 I + \delta_2 H - \mu R \end{aligned}$$

where, S', E', I', H', R' are the rate of change of Susceptible (S), Exposed (E), Infective (I), Hospitalised (H) and Recovered (R). The compartment S contains individual who are all susceptible. Likewise, E contains individual who are exposed to the infected person(s) and I contains individual who are infected by the disease. Also, H fills the compartment with person who are infected at high risk and hospitalised. Whereas, compartment R consists of recovered individuals. The other parameters are described in the table below,

Table 1 Parameter Description of the model

Parameter	Description	Parameter	Description
Λ	Recruitment rate	δ_1	Rate at which I recovers
β_1	Infection rate from S to I	δ_2	Rate at which H recovers
β_2	Infection rate from S to E	k_1	Disease caused death rate at I
γ	Rate at which E moves to I	k_2	Disease caused death rate at H
λ	Rate at which E moves to R	μ	Natural mortality
η	Rate at which I moves to H	α_1	Saturated incidence rate

The concise explanation of the biological significance of each rate is provided below:

Clearly, Λ determines the rate at which new susceptible individuals enter the population, impacting disease spread dynamics and the size of the susceptible pool. β_1, reflects the likelihood of transmission per contact between susceptible and infectious individuals, crucial for estimating new infections. β_2, indicates the probability of susceptible individuals becoming exposed to the pathogen, accounting for latent or asymptomatic transmission. γ, represents the speed at which exposed individuals become infectious, influencing the timing and intensity of disease transmission. λ, determines the likelihood of exposed individuals recovering without becoming infectious, impacting overall disease outcomes and herd immunity. η, reflects the likelihood of severe cases requiring hospitalization, essential for healthcare resource planning and mitigating disease impact on morbidity and mortality. δ_1, represents the speed at which infectious individuals recover from the disease, influencing the duration of infectiousness and overall disease transmission dynamics. δ_2, determines the speed at which hospitalized individuals recover from the disease, impacting healthcare resource utilization and patient outcomes. k_1 and k_2, indicates the likelihood of death among the infectious and hospitalised individuals due to the disease, respectively. These provide an insight into disease severity and mortality risk during infection. μ, represents the background rate of death in the population unrelated to the disease, providing context for interpreting disease-specific mortality rates and overall mortality burden. Finally α_1, marks the point at which disease transmission reaches a stabilizing threshold due to factors like immunity or limited susceptible individuals, informing projections of epidemic trajectories and potential control measures.

2.2. Preliminary Lemmas:

2.2.1. Lemma 1

Prove that the solution set of system (1) are positive for all $t > 0$.

Proof.

We have,

$$\frac{dS}{dt} = \Lambda - \frac{\beta_1 SI}{1+\alpha_1 I} - \beta_2 SI - \mu S$$

$$\geq -\left(\frac{\beta_1 I}{1+\alpha_1 I} + \beta_2 I + \mu\right) S$$

$$\frac{dS}{S} \geq -\left(\frac{\beta_1 I}{1+\alpha_1 I} + \beta_2 I + \mu\right) dt$$

Integrating and solving, we have

$$\log S \geq -\left(\frac{\beta_1 I}{1+\alpha_1 I} + \beta_2 I + \mu\right)t + \log C$$

$$\frac{\log S}{\log C} \geq -\left(\frac{\beta_1 I}{1+\alpha_1 I} + \beta_2 I + \mu\right)t$$

Raising to exponentials and simplifying, we get

$S(t) \geq 0$

Similarly, we can show that $E(t) \geq 0,\ I(t) \geq 0,\ H(t) \geq 0$ and $R(t) \geq 0.$

Thus, for all $t > 0$ the proposed system (1) is positive.

2.2.2. Lemma 2

Show that the solution of system (1) along with the initial conditions are bounded in the region $\Phi \subset \mathbb{R}^5_+$, where $\Phi = \left\{\left(S(t), E(t), I(t), H(t), R(t)\right) \in \mathbb{R}^5_+ : N(t) \leq \frac{\Lambda}{\mu}\right\}$.

Proof.

Summing up all the equations of (1) we get

$$\begin{aligned} N' &= S' + E' + I' + H' + R' \\ &\leq \Lambda - \mu\left(S + E + I + H + R\right). \end{aligned}$$

$$\frac{dN(t)}{dt} \leq \Lambda - \mu N$$

Thus,

$$\limsup_{t\to\infty} N(t) \leq \frac{\Lambda}{\mu}.$$

Hence, the solution set is bounded within the region Φ.

2.3. Determination of Reproduction number R_0

Reproduction number denoted as R_0 holds significant importance in epidemiology, serving as an indicator of the average number of secondary instances of an infectious disease that an infected individual is anticipated to generate within a population entirely susceptible to the illness. It plays a pivotal role in assisting public health officials in gauging the potential risk of disease transmission.

If R_0 exceeds 1, it suggests a likelihood of the disease spreading among individuals and triggering an outbreak. Conversely, if R_0 is less than 1, the disease is less likely to proliferate extensively and may eventually fade out.

R_0 may change based on the contagiousness of the virus, population behaviour and treatments like immunisation or social isolation.

The R_0 value of the proposed model can be determined by finding the Jacobian matrix of system (1) as follows,

$$J(E,I,H)=\begin{pmatrix} \beta_2 S-(\gamma+\lambda+\mu) & 0 & 0 \\ \gamma & \dfrac{\beta_1 S}{(1+\alpha_1 I)^2}-(\eta+\delta_1+k_1+\mu) & 0 \\ 0 & \eta & -(\delta_2+k_2+\mu) \end{pmatrix}.$$

The transmission and the transition matrix is given as

$$F=\begin{pmatrix} \beta_2 S & 0 & 0 \\ 0 & \dfrac{\beta_1 S}{(1+\alpha_1 I)^2} & 0 \\ 0 & 0 & 0 \end{pmatrix}. \quad \text{and} \quad V=\begin{pmatrix} -(\gamma+\lambda+\mu) & 0 & 0 \\ \gamma & -(\eta+\delta_1+k_1+\mu) & 0 \\ 0 & \eta & -(\delta_2+k_2+\mu) \end{pmatrix}.$$

Thus,

$$FV^{-1}=\begin{pmatrix} \dfrac{-1}{(\gamma+\lambda+\mu)} & 0 & 0 \\ \dfrac{\gamma}{(\gamma+\lambda+\mu)(\eta+\delta_1+k_1+\mu)} & \dfrac{-1}{(\eta+\delta_1+k_1+\mu)} & 0 \\ \dfrac{-\gamma\eta}{(\gamma+\lambda+\mu)(\eta+\delta_1+k_1+\mu)(\delta_2+k_2+\mu)} & \dfrac{\eta}{(\eta+\delta_1+k_1+\mu)(\delta_2+k_2+\mu)} & \dfrac{-1}{(\delta_2+k_2+\mu)} \end{pmatrix}.$$

The eigenvalues of the above matrix are $0,\ \dfrac{-\beta_2 S}{(\gamma+\lambda+\mu)},\ \dfrac{-\beta_1 S}{(1+\alpha_1 I)^2(\eta+\delta_1+k_1+\mu)}$.

Thus, $R_0=\max\{R_1,R_2\}$, where $R_1=\dfrac{\Lambda\beta_2}{\mu(\gamma+\lambda+\mu)}$ and $R_2=\dfrac{\Lambda\beta_1}{\mu(\eta+\delta_1+k_1+\mu)}$ .....(2)

2.4. Existence of Equilibrium points

Equilibrium points play a vital role in dissecting infection trends within a population in the realm of epidemiology. They represent stable situations where the prevalence of an infectious disease remains constant over time. Understanding these points is pivotal in deciphering the dynamics of epidemics and devising effective strategies for disease management and prevention.

Two primary equilibrium points exist: the Disease-Free Equilibrium (DFE) and the Endemic Equilibrium (EE). At the DFE, the population is devoid of any illness. The onset of an epidemic typically occurs when a contagious disease infiltrates a susceptible population. Conversely, the EE emerges when the level of infection within the population stabilizes. This equilibrium signifies a scenario where the disease either persists continually throughout the population over time or establishes an endemic presence.

The DFE of system (1) is considered to be $(S_0,E_0,I_0,H_0,R_0)=(1,0,0,0,0)$ and the EE can be obtained by simplifying every equation of system (1) as follows:

$$S^*=\frac{\Lambda\beta_2(1+\alpha_1 I^*)(\gamma+\lambda+\mu)}{\beta_1\beta_2(\gamma+\lambda+\mu)I^*+\beta_2(1+\alpha_1 I^*)\left[\Lambda\beta_2(1+\alpha_1 I^*)-(\gamma+\lambda+\mu)\left(\beta_1 I^*+(1+\alpha_1 I^*)\mu\right)\right]+\mu\beta_2(1+\alpha_1 I^*)(\gamma+\lambda+\mu)}.$$

$$E^*=\frac{\Lambda\beta_2(1+\alpha_1 I^*)-(\gamma+\lambda+\mu)\left(\beta_1 I^*+(1+\alpha_1 I^*)\mu\right)}{(\gamma+\lambda+\mu)\beta_2}.$$

$$H^* = \frac{\eta I^*}{\delta_2 + k_2 + \mu}.$$

$$R^* = \frac{\Lambda\beta_2(\delta_2 + k_2 + \mu)\lambda(1+\alpha_1 I^*) - \lambda(\delta_2 + k_2 + \mu)(\gamma + \lambda + \mu)\left[\beta_1 I^* + (1+\alpha_1 I^*)\mu\right] + \delta_1\beta_2(\delta_2 + k_2 + \mu)(\gamma + \lambda + \mu)I^* + \eta\delta_2\beta_2(\gamma + \lambda + \mu)I^*}{\beta\mu(\delta_2 + k_2 + \mu)(\gamma + \lambda + \mu)}$$

The value of I^* can be obtained by solving the below cubic equation

$$\left(A_1K_3 - A_1D_2K_4\right)I^{*3} + \left(A_1K_2 + A_2K_3 - A_2D_2K_4\right)I^{*2} + \left(K_1 + A_3K_3 + A_2K_2 - A_3D_2K_4\right)I^* + A_3K_2 = 0.$$

……..(3)

Where,

$$\begin{aligned} D_1 = &\left[\Lambda\alpha_1^2\beta_2^2 - \alpha_1\beta_1\beta_2(\gamma + \lambda + \mu) - \alpha_1^2\beta_2\mu(\gamma + \lambda + \mu)\right]I^{*2} \\ &+\left[2\Lambda\alpha_1\beta_2^2 - \beta_1\beta_2(\gamma + \lambda + \mu) - 2\alpha_1\beta_2\mu(\gamma + \lambda + \mu)\right]I^* \\ &+\left[\Lambda\beta_2^2 + \beta_1\beta_2(\gamma + \lambda + \mu)\right] \end{aligned}$$

$$D_2 = \beta_2(\gamma + \lambda + \mu)$$

$$\begin{aligned} K_1 &= \Lambda\beta_1\beta_2^2(\gamma + \lambda + \mu)^2, \\ K_2 &= \Lambda\gamma\beta_2 - \mu\gamma(\gamma + \lambda + \mu), \\ K_3 &= \Lambda\gamma\beta_2\alpha_1 - \gamma(\gamma + \lambda + \mu)\beta_1 - \gamma\mu(\gamma + \lambda + \mu)\alpha_1, \\ K_4 &= \eta + \delta_1 + k_1 + \mu \end{aligned}.$$

$$\begin{aligned} A_1 &= \Lambda\alpha_1^2\beta_2^2 - \alpha_1\beta_1\beta_2(\gamma + \lambda + \mu) - \alpha_1^2\mu\beta_2(\gamma + \lambda + \mu), \\ A_2 &= 2\Lambda\alpha_1\beta_2^2 - \beta_1\beta_2(\gamma + \lambda + \mu) - 2\alpha_1\mu\beta_2(\gamma + \lambda + \mu), \\ A_3 &= \Lambda\beta_2 + \beta_1\beta_2(\gamma + \lambda + \mu) \end{aligned}$$

Using Routh-Hurwitz criterion we show that equation (3) is stable by the following condition.

- $\frac{A_1K_2 + A_2K_3 - A_2D_2K_4}{A_1K_3 - A_1D_2K_4} > 0$
- $\frac{A_3K_2}{A_1K_3 - A_1D_2K_4} > 0$ and
- $J_1J_2 - J_0 > 0$

Where,

$$J_0 = \frac{A_3K_2}{A_1K_3 - A_1D_2K_4},\ J_1 = \frac{K_1 + A_2K_2 + A_3K_3 - A_3D_2K_4}{A_1K_3 - A_1D_2K_4} \text{ and } J_2 = \frac{A_1K_2 + A_2K_3 - A_2D_2K_4}{A_1K_3 - A_1D_2K_4}.$$

2.5. Stability Analysis of the Equilibrium points:

Conducting a stability assessment of the Disease-Free Equilibrium (DFE) involves examining whether the population is susceptible to disease outbreaks or remains immune under specific conditions. Unravelling the intricacies of disease

transmission mechanisms and predicting whether the disease will persist or fade away necessitates a thorough stability analysis of the Endemic Equilibrium (EE). By scrutinizing the stability of both disease-free and endemic equilibria in mathematical epidemiology models, researchers and public health experts gain insights into the long-term dynamics of infectious diseases. Understanding stability criteria allows for prognostications regarding the disappearance, sustained prevalence, or fluctuation patterns of diseases within a population, thereby facilitating the development of effective management and prevention strategies.

The stability of these equilibria can be ascertained through specific theorems and proofs, as below:

2.5.1. Theorem 1

If the value of $R_0 < 1$, the system (1) at DFE is locally asymptotically stable, whereas it is unstable when $R_0 > 1$

Proof

From (1), it is observed that the first four equations are independent of R. So, considering only those four equations, the stability of system (1) is determined.

Let $M_1 = (S_0, E_0, I_0, H_0)$ be the system of equations at DFE. The Jacobian matrix of M_1 is obtained as below

$$J(M_1) = \begin{pmatrix} -\mu & -\beta_2 S_0 & -\beta_1 S_0 & 0 \\ 0 & \beta_2 S_0 - (\gamma + \lambda + \mu) & 0 & 0 \\ 0 & \gamma & \beta_1 S_0 - (\eta + \delta_1 + k_1 + \mu) & 0 \\ 0 & 0 & \eta & -(\delta_2 + k_2 + \mu) \end{pmatrix}$$

The above matrix has two obvious negative eigenvalues: $-\mu$ and $-(\delta_2 + k_2 + \mu)$.

Thus, the matrix $J(M_1)$ reduces as below submatrix

$$J_s(M_1) = \begin{pmatrix} \beta_2 S_0 - (\gamma + \lambda + \mu) & 0 \\ \gamma & \beta_1 S_0 - (\eta + \delta_1 + k_1 + \mu) \end{pmatrix}$$

From the corollary of Gershgorin's Circle Theorem, it satisfies the below conditions,

- $\beta_2 S_0 - (\gamma + \lambda + \mu) < 0$
- $\beta_1 S_0 - (\eta + \delta_1 + k_1 + \mu) < -\gamma$

Simplifying the first condition, we get

$$\frac{\beta_2 S_0}{(\gamma + \lambda + \mu)} < 1 \Rightarrow R_1 < 1. \qquad \text{.....(4)}$$

Also, the second condition implies $\beta_1 S_0 - (\eta + \delta_1 + k_1 + \mu) < 0$ since the negative of γ is less than zero.

$$\therefore \frac{\beta_1 S_0}{(\eta + \lambda + k_1 + \mu)} < 1 \Rightarrow R_2 < 1. \text{ .....(5)}$$

Hence, from (4) and (5) it is shown that the DFE is locally asymptotically stable as $R_0 < 1$.

2.5.2. Theorem 2

For $R_0 > 1$, the system (1) is locally asymptotically stable at Endemic Equilibrium.

Proof.

Let $M_2 = \left(S^*, E^*, I^*, H^*\right)$ be the system at endemic equilibrium. The Jacobian matrix of M_2 is obtained as

$$J(M_2) = \begin{pmatrix} \frac{\beta_1 I^*}{1+\alpha_1 I^*} - \beta_2 E^* - \mu & -\beta_2 S^* & \frac{\beta_1 S^*}{\left(1+\alpha_1 I^*\right)^2} & 0 \\ \beta_2 E^* & \beta_2 S^* - (\gamma+\lambda+\mu) & 0 & 0 \\ \frac{\beta_1 I^*}{1+\alpha_1 I^*} & \gamma & \frac{\beta_1 S^*}{\left(1+\alpha_1 I^*\right)^2} - (\eta+\delta_1+k_1+\mu) & 0 \\ 0 & 0 & \eta & -(\delta_2+k_2+\mu) \end{pmatrix}$$

Thus, $-(\delta_2 + k_2 + \mu)$ is one of the eigenvalue and it is negative.

Thus, the matrix $J(M_2)$ reduces as below submatrix

$$J_s(M_2) = \begin{pmatrix} \frac{\beta_1 I^*}{1+\alpha_1 I^*} - \beta_2 E^* - \mu & -\beta_2 S^* & \frac{\beta_1 S^*}{\left(1+\alpha_1 I^*\right)^2} \\ \beta_2 E^* & \beta_2 S^* - (\gamma+\lambda+\mu) & 0 \\ \frac{\beta_1 I^*}{1+\alpha_1 I^*} & \gamma & \frac{\beta_1 S^*}{\left(1+\alpha_1 I^*\right)^2} - (\eta+\delta_1+k_1+\mu) \end{pmatrix}$$

From the corollary of Gershgorin's Circle Theorem, it satisfies the below conditions,

$$\frac{\beta_1 I^*}{1+\alpha_1 I^*} - \beta_2 E^* - \mu < -\left(\beta_2 S^* + \frac{\beta_1 S^*}{\left(1+\alpha_1 I^*\right)^2}\right) \quad \text{.....(6)}$$

$$\beta_2 S^* - (\gamma+\lambda+\mu) < -\beta_2 E^* \quad \text{.....(7)}$$

$$\frac{\beta_1 S^*}{\left(1+\alpha_1 I^*\right)^2} - (\eta+\delta_1+k_1+\mu) < -\left(\frac{\beta_1 I^*}{1+\alpha_1 I^*} + \gamma\right) \text{.....(8)}$$

Simplifying (7), we get

$$-\frac{(\gamma+\lambda+\mu)}{\beta_2 E^*}(R_1 - 1) < 1$$

$$\Rightarrow -\frac{(\gamma+\lambda+\mu)}{\beta_2 E^*}\left(R_1 - 1\right) < 0$$

$$\Rightarrow -\left(R_1 - 1\right) < 0$$

Thus, $R_1 > 1$ .....(9)

Similarly solving (8), we get

$$-\frac{(\eta+\delta_1+k_1+\mu)}{\beta_1 I^* + \gamma(1+\alpha_1 I^*)}\left(R_2 - 1 - \alpha_1 I^*\right) < 1$$

$$\Rightarrow -(R_2 - 1) < 1 - \alpha_1 I^*$$
$$\Rightarrow -(R_2 - 1) < 0$$

Hence, $R_2 > 1$ .....(10)

From (9) and (10), it is clear that the EE is locally asymptotically stable as $R_0 > 1$.

3. Results and Discussion

To grasp the intricacies of infectious disease transmission dynamics, numerical simulations are employed. This entails utilizing the numerical parameters outlined in (Zhang et al., 2014) for analysis, as follows:

$$\Lambda = 10,\ \beta_1 = \beta_2 = 0.05,\ \alpha_1 = 0.8,\ \gamma = 1.2,\ \lambda = \delta_1 = \delta_2 = 0.4,\ \eta = 0.1,\ k_1 = k_2 = 0.2,\ \mu = 0.5.$$

Also, setting initial values as $S(0) = 100,\ E(0) = 3,\ I(0) = 1,\ H(0) = 0,\ R(0) = 0$ we obtain dynamics of the proposed model as given in the below figure.

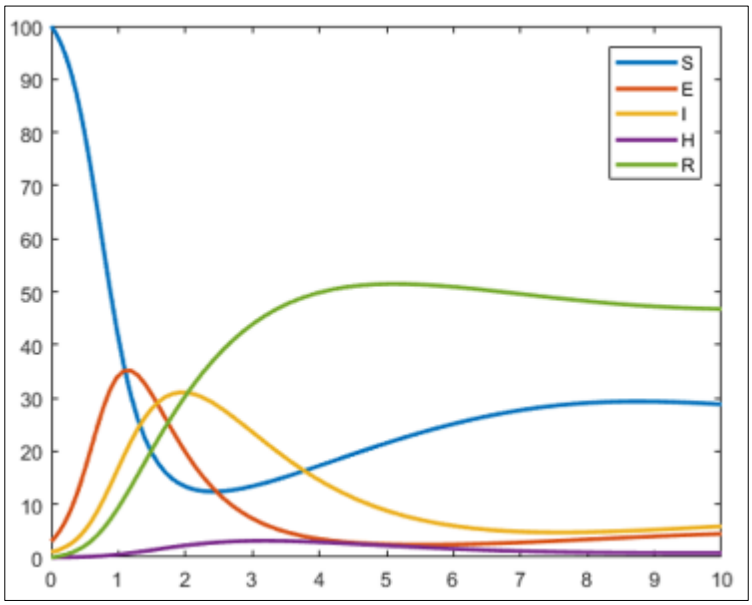

Figure 1 Representation of Dynamical behaviour of the proposed model

The model's analysis is depicted in the graphical representation above. Observing the graph, a notable decline is evident among those susceptible to the disease. The curves representing exposed and infected individuals show no significant increase, and if there is any uptick, it's short-lived. Furthermore, the constrained nature of the curve representing hospitalized individuals suggests efficient management and provision of adequate facilities for those requiring admission. As per the model's implications, the recovery curve displays a promising trend, signalling a favourable outcome. Moreover, the recovery curve consistently maintains its positive trajectory.

The robustness of the model is illustrated in the figure below, showcasing the effectiveness of the incidence rate in ensuring the availability of hospital resources. This reaffirms the accessibility of hospital facilities for all infected individuals.

Hence, the preceding figures illustrate a rapid decline in the susceptible population, minimal increases in exposed and infectious cases, efficient management of hospitalizations, and an encouragingly sustained recovery rate. This portrayal hints at a favourable and effectively managed scenario within the scope of the proposed model.

Moreover, the stability of the proposed model can be visually confirmed through graphical verification using MATLAB, yielding the following output:

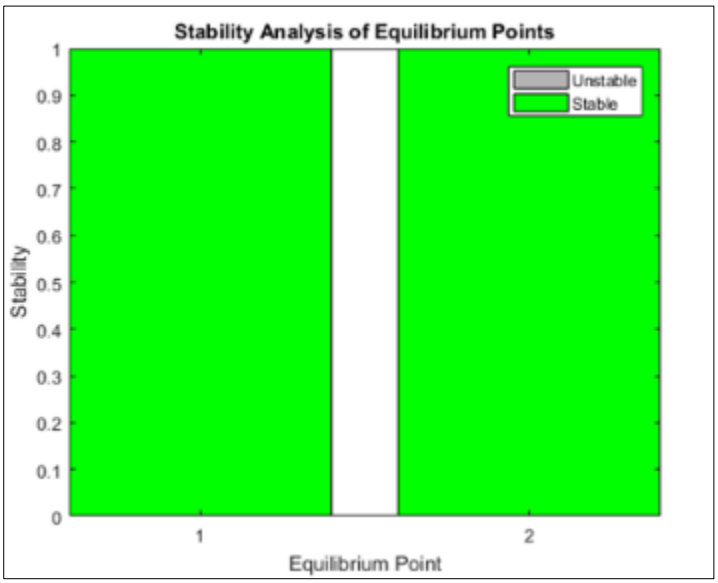

Figure 2 Graphical representation of the stability of equilibrium points

In Figure 2, the Equilibrium point 1 and 2 represents the Disease-free equilibrium and the Endemic Equilibrium point respectively.

To determine the numerical value of basic reproduction number, the considered value is substituted in (2).

Equation (2) gives, $R_0 = \max\{R_1, R_2\}$ where

$$R_1 = \frac{\Lambda\beta_2}{\mu(\gamma+\lambda+\mu)} = \frac{10\times 0.05}{0.5\times(1.2+0.4+0.5)} = 0.4762$$

$$R_2 = \frac{\Lambda\beta_1}{\mu(\eta+\delta_1+k_1+\mu)} = \frac{10\times 0.05}{0.5\times(0.1+0.4+0.2+0.5)} = 0.8333$$

Clearly this shows that

$$R_0 = 0.8333 \qquad \text{.....(11)}$$

3.1. Sensitivity Analysis

Conducting sensitivity analysis is indispensable for comprehending the impacts of individual parameters in disease transmission models. It facilitates experiment design, data assimilation, and the simplification of intricate models by assessing how resilient predictions are to variations in parameter values and inherent errors. The basic reproduction number, a pivotal epidemiological metric, often garners attention among disease transmission model parameters.

Identifying these foundational factors is crucial for effective intervention planning. Sensitivity indices, such as the normalized forward sensitivity index, quantify the effect of parameter alterations on model outcomes. This index delineates the correlation between the relative change in a model output and the relative change in a specific parameter, particularly beneficial for differentiable variables. By pinpointing key parameters through sensitivity indices,

researchers can concentrate their endeavours on enhancing precision and obtaining accurate results. Consequently, the model becomes more reliable in guiding public health decisions and interventions.

From equation (2) and (11), it is clear that the expression of R_0 is $\frac{\Lambda\beta_1}{\mu(\eta+\delta_1+k_1+\mu)}$. The sensitivity indices for the same parameter values considered earlier, by the definition of normalised forward sensitivity index of R_0, the latter simulations are performed.

$$\begin{aligned}\Gamma_{\mu}^{R_0} &= \frac{-\Lambda\beta_1(\eta+\delta_1+k_1+2\mu)}{\mu^2(\eta+\delta_1+k_1+\mu)^2}\times\frac{\mu^2(\eta+\delta_1+k_1+\mu)}{\Lambda\beta_1}\\ &= -\frac{(\eta+\delta_1+k_1+2\mu)}{(\eta+\delta_1+k_1+\mu)}\\ &= -1.4167\end{aligned}$$

Similarly, the sensitivity indices for other parametric values is obtained as given in Table 2.

Table 2 Sensitivity indices of R_0

Parameter	Sensitivity Index
μ	–1.4167
η	–0.0833
δ_1	–0.3333
k_1	–0.1667
Λ	+1
β_1	+1

From Table 2, it is clear that the most negative sensitive parameter to the reproduction number of the proposed model is μ. And the most positive sensitive parameter is the recruitment rate, Λ and the β_1. Thus, increasing the value of μ by 10%, will decrease the basic reproduction number R_0 by 14.167 %. Similarly, for increase in other parameters the basic reproduction number R_0 will decrease by its corresponding percentage.

4. Conclusion

The incidence rate holds paramount importance in assessing the force of infection as the disease disseminates among the susceptible population. Additionally, it aids in gauging the inhibitory impact induced by behavioural alterations in susceptible individuals. The model's positivity and boundedness have been confirmed, ensuring its reliability. Subsequently, the calculation of the basic reproduction number unveils two distinct scenarios. This leads to the identification of equilibrium points, namely the Disease-Free Equilibrium (DFE) and the Endemic Equilibrium (EE). The numerical value of R_0 is less than one for a locally stable DFE, while the numerical value of R_0 is greater than one for the endemic equilibrium to be locally stable. Thorough scrutiny is applied to the evidence bolstering the initial assertion. The numerical simulation commences with a graphical depiction, shedding light on the suggested model's dynamic behaviour. The results unveil an optimistic outlook, characterized by a heightened recovery curve juxtaposed with a diminished infective curve. Consequently, the proposed model solidifies its standing as a robust epidemiological framework, significantly enriching our understanding of infectious dynamics.

Compliance with ethical standards

Disclosure of conflict of interest

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