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Mathematical model of a SCIR epidemic system with migration and nonlinear incidence function

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Abstract

In this paper, we proposed a generalization for a model that considers Susceptible, Infected, Carrier, and Recovered by introducing a general incidence rate and considering migration in all its populations. This model has the characteristic that carriers and infected can transmit the disease, besides it has not a disease-free equilibrium point and no basic reproductive number. The focus of this study is to show a generalized model and the conditions required to analyze the equilibrium point stability. Using an appropriate Lyapunov function and with suitable conditions on the functions involved in the general incidence, we showed that the disease equilibrium point is globally asymptotically stable. Also, we presented numerical simulations of two applications to illustrate the results obtained from the analytical part.

Keywords: Global stability, migration, Lypunov function, non-linear incidence rate.

2020 MSC: 34D20, 34D23, 92D30.

1. Introduction

In disease transmission, there are many influential factors in its dynamics, for example, the ability of pathogens to infect and replicate within a host [1], the mode of transmission can vary, some diseases are spread through respiratory droplets while others spread through direct contact with fluids of infected person [12], age, immune system status and genetic predisposition of host [3, 19], behavioral factors as poor hygiene, not wearing mask, engaging in close contacts with others [29], environmental factors as high population density, poor sanitation [27], migration and immigration [28], between others. Regarding factors such as migration or immigration of people, this has a great influence on the transmission of a disease, especially in those diseases where asymptomatic or carriers can spread or transmit the disease, since they can not be detected or isolated unless there is a traceability of contacts with symptomatic [4, 6, 9, 10, 16, 18, 20, 23]. Therefore, it is vital to include migration processes in mathematical models to provide a more comprehensive and accurate understanding of the world around us, and to inform better decision-making for individuals, organizations, and policymakers. In this sense, we propose a novel model that accounts for migration across populations categorized as susceptible, carrier or asymptomatic,

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infected, and recovered. In this model, carriers are able to transmit the disease using a general nonlinear incidence rate represented by $S[\beta C + h(I)]$, where the function h is increasing and concave, reaching saturation for large values of I. The incidence rate for infected individuals is chosen to be of this form ($Sh(I)$) as the identification of infected individuals is primarily through the manifestation of symptoms, making it easier to isolate and treat them. This identification process can reduce transmission rates. Asymptomatic carriers, on the other hand, are not easily identified unless contact tracing is performed. Thus, the model employs a bilinear incidence rate ($S\beta C$) for asymptomatic individuals.

There are mathematical models considering migration [14, 22, 25, 26], where the effect that migration or immigration can have on the transmission of a disease is studied, but few mathematical models consider migration and nonlinear incidence rates, since nonlinearities make it difficult to easily find equilibrium points. Moreover, proving that the equilibrium points are asymptotically stable can be even more challenging. An earlier work has included immigration and non linear incidence rate of the form $f(S, I)$ in a SEIR model [17]. An SEI model with migration and incidence of the form $Sf(I)$ was studied in [24]. Global dynamics was studied in a SEIR model with immigration and incidence rate of the form $C(N)\frac{SI}{N}$, where $C(N)$ is a function that depends on the population size N [31]. A SEIR type age-structured model with migration and nonlinear incidence rates was studied in [32]. While models where asymptomatic people transmit the disease and consider non-linear incidence rates but do not consider immigration have been studied in [7, 8, 21].

The aim of this paper is to prove the global stability of equilibriums of a $SCIR$ type model. The organization of this paper is as follows. In Section 2 we described the model and found a positively invariant set. The existence of unique equilibrium point is studied in Section 3. In Section 4 The global stability of disease equilibrium point is proved using the Lyapunov direct method. In Section 5 we give some particular nonlinear incidence rates to illustrate by numerical simulations our main results. In Section 6 we discuss our results.

2. Model formulation

We consider an infectious disease for which the population consists of susceptibles, carriers or asymptomatics, infectives individuals, and recovered, with the sizes of the groups given by the variables S, C, I, and R, respectively. We assume that there is constant recruitment given by birth or migration process into each of the four class at rate k_1, k_2, k_3, and k_4. The rate constant for nature death is μ_1. A susceptible person become infected carrier by the contact with a asymptomatic or an infected person. We assume that the incidence rate is nonlinear of the form $S[\beta C + h(I)]$ and that the function h satisfies

I. $h(I) > 0$, and $h(0) = 0$;
II. $h'(I) \geqslant 0$;
III. $h''(I) < 0$.

A newly infected individual can show symptoms with rate γq or can recovered without show symptoms at rate $\gamma(1-q)$. The death rate for Carriers, Infected, and Recovered are μ_2, μ_3, and μ_4, respectively. Infected individuals (I), have an average recovery time of $1/p$. The system of differential equations for the model is

$$\begin{aligned} \frac{dS}{dt} &= k_1 - S[\beta C + h(I)] - \mu_1 S, & \frac{dC}{dt} &= k_2 + S[\beta C + h(I)] - \gamma C - \mu_2 C, \\ \frac{dI}{dt} &= k_3 + \gamma q C - pI - \mu_3 I, & \frac{dR}{dt} &= k_4 + \gamma(1-q)C + pI - \mu_4 R. \end{aligned} \tag{2.1}$$

All the parameters are positive constants and $0 < q < 1$. Let $k = k_1 + k_2 + k_3 + k_4$, $\mu = \min\{\mu_1, \mu_2, \mu_3, \mu_4\}$ and $\Omega = \{(S, C, I, R) \in \mathbb{R}^4_{\geqslant 0} : 0 < S + C + I + R \leqslant \frac{k}{\mu}\}$.

Proposition 2.1. *The set Ω is positively invariant.*

Proof. First, we will show that the solutions of system (2.1) are greater than or equal to zero. Since $\frac{dS}{dt}\Big|_{S=0} = k_1 > 0$, $\frac{dC}{dt}\Big|_{C=0} = k_2 + Sh(I) > 0$, $\frac{dI}{dt}\Big|_{I=0} = k_3 + \gamma qC > 0$, $\frac{dR}{dt}\Big|_{R=0} = k_4 + \gamma(1-q)C + pI > 0$ within $\mathbb{R}^4_{\geqslant 0}$, then by proposition 2.1 of [11], the set $\mathbb{R}^4_{\geqslant 0}$ is positive invariant and in this way all the solutions are greater than or equal to zero. Finally, we will show that the set omega is positively invariant. Let $N = S + C + I + R$. Then $\frac{dN}{dt} = k_1 + k_2 + k_3 + k_4 - \mu_1 S - \mu_2 C - \mu_3 I - \mu_4 R \leqslant k - \mu N$. It follows that $\frac{dN}{dt} \leqslant 0$ if $N \geqslant \frac{k}{\mu}$. Besides, we have,

$$N \leqslant \frac{k}{\mu} + \left(N(0) - \frac{k}{\mu}\right) e^{-\mu t}, \text{ for all, } t \geqslant 0.$$

In particular, $N \leqslant \frac{k}{\mu}$ if $N(0) \leqslant \frac{k}{\mu}$. Therefore, the set Ω is positively invariant. In addition, if $N(0) \geqslant \frac{k}{\mu}$, then either the solutions enters Ω infinite time or $\Omega(t)$ approaches $\frac{k}{\mu}$ asymptotically. Hence, the set Ω attracts all solutions in $\mathbb{R}^4_{\geqslant 0}$. □

3. Equilibrium points

Equilibria for the system (2.1) can be found by setting the right sides of the four differential equations of (2.1) equal to zero, giving the algebraic system,

$$k_1 - S[\beta C + h(I)] - \mu_1 S = 0, \tag{3.1}$$
$$k_2 + S[\beta C + h(I)] - \gamma C - \mu_2 C = 0, \tag{3.2}$$
$$k_3 + \gamma qC - pI - \mu_3 I = 0, \tag{3.3}$$
$$k_4 + \gamma(1-q)C + pI - \mu_4 R = 0.$$

from equation (3.3) we have

$$C = \frac{(p+\mu_3)I - k_3}{\gamma q},$$

In order to obtain $C > 0$, we require $I > \frac{k_3}{(p+\mu_3)}$. From equation (3.1) and (3.2) we have

$$S[\beta C + h(I)] = k_1 - \mu_1 S = \gamma C + \mu_2 C - k_2.$$

In this way, S can be written in the form

$$S = \frac{k_1 + k_2 - (\gamma + \mu_2)C}{\mu_1} = \frac{(k_1+k_2)\gamma q - (\gamma+\mu_2)[(p+\mu_3)I - k_3]}{\gamma q \mu_1}.$$

To have $S > 0$, we need $I < \hat{I} = \frac{\gamma q(k_1+k_2) + k_3(\gamma+\mu_2)}{(p+\mu_3)(\gamma+\mu_2)}$. Now, note that the equation (3.1) can be rewritten as

$$-S\left([\beta C + h(I)] + \mu_1 - \frac{k_1}{S}\right) = 0,$$

which is equivalent to

$$SF(I) = 0,$$

where

$$F(I) = \frac{\beta[(p+\mu_3)I - k_3]}{\gamma q} + h(I) + \mu_1 - \mu_1 \frac{k_1 \gamma q}{(k_1+k_2)\gamma q - (\gamma+\mu_2)[(p+\mu_3)I - k_3]}.$$

If we solve $F(I) = 0$ for I in the interval $\left(\frac{k_3}{p+\mu_3}, \hat{I}\right)$, we found the equilibrium points of the model. We see that

$$F\left(\frac{k_3}{p+\mu_3}\right) = h\left(\frac{k_3}{p+\mu_3}\right) + \mu_1 - \mu_1 \frac{k_1}{k_1+k_2} > 0.$$

Besides, $F(I)$ tends to $-\infty$ when I tends to $\hat{I}$, by the continuity of F we have that there exists a zero of F in $\left(\frac{k_3}{p+\mu_3}, \hat{I}\right)$. Now we show that this zero is unique, to do this we found the second derivative of F and see that

$$F''(I) = h''(I) - \frac{2\mu_1 k_1 \gamma q(\gamma+\mu_2)^2(p+\mu_3)^2}{\{\gamma q(k_1+k_2) - (\gamma+\mu_2)[(p+\mu_3)I - k_3]\}^3} < 0.$$

Then F is concave in $(\frac{k_3}{p+\mu_3}, \hat{I})$, this implies that the zero is unique. We resume the above analysis in the next proposition.

Proposition 3.1. *There exists a unique equilibrium point* (S^*, C^*, I^*, R^*).

Remark 3.2. We clarify that there is no point free of infection, because we have $\left(\frac{dC}{dt} + \frac{dI}{dt}\right)_{C=I=0} = k_2 + k_3 > 0$.

4. Stability analysis

In the next analysis, we omit the equation for recovery state, R, because it does not appear in the other equations.

Theorem 4.1 (The equilibrium point is GAS)**.** *The equilibrium point is globally asymptotically stable.*

Proof. Let be L the function defined by

$$L = (S - S^* \ln S) + (C - C^* \ln C) + a\left(I - h(I^*)\int_{I^*}^{I} \frac{d\tau}{h(\tau)}\right),$$

where $a = \frac{S^* h(I^*)}{\gamma q C^*}$ .Taking the derivative of function L along trajectories of system (2.1), we have

$$\dot{L} = \left(1 - \frac{S^*}{S}\right)\dot{S} + \left(1 - \frac{C^*}{C}\right)\dot{C} + a\left(1 - \frac{h(I^*)}{h(I)}\right)\dot{I}.$$

This is equivalent to

$$\begin{aligned}\dot{L} = &\left(1 - \frac{S^*}{S}\right)(k_1 - S[\beta C + h(I)] - \mu_1 S) + \left(1 - \frac{C^*}{C}\right)(k_2 + S[\beta C + h(I)] - \gamma C - \mu_2 C)\\ &+ a\left(1 - \frac{h(I^*)}{h(I)}\right)(k_3 + \gamma q C - pI - \mu_3 I).\end{aligned}$$

Using the equations

$$k_1 = S^*[\beta C^* + h(I^*)] + \mu_1 S^*, \qquad \gamma + \mu_2 = \frac{k_2 + S^*[\beta C^* + h(I^*)]}{C^*}, \qquad p + \mu_3 = \frac{k_3 + \gamma q C^*}{I^*},$$

we have

$$\begin{aligned}\dot{L} = &\left(1 - \frac{S^*}{S}\right)(S^*[\beta C^* + h(I^*)] + \mu_1 S^* - S[\beta C + h(I)] - \mu_1 S)\\ &+ \left(1 - \frac{C^*}{C}\right)(k_2 + S[\beta C + h(I)] - \frac{k_2 + S^*[\beta C^* + h(I^*)]}{C^*} C)\\ &+ a\left(1 - \frac{h(I^*)}{h(I)}\right)(k_3 + \gamma q C - \frac{k_3 + \gamma q C^*}{I^*} I)\\ = &\left(1 - \frac{S^*}{S}\right)\left(S^*[\beta C^* + h(I^*)](1 - \frac{S[\beta C + h(I)]}{S^*[\beta C^* + h(I^*)]}) + \mu_1 S^*(1 - \frac{S}{S^*})\right)\end{aligned}$$

$$+\left(1-\frac{C^*}{C}\right)\left(k_2\left(1-\frac{C}{C^*}\right)+S^*\left[\beta C^*+h(I^*)\right]\left(\frac{S\left[\beta C+h(I)\right]}{S^*\left[\beta C^*+h(I^*)\right]}-\frac{C}{C^*}\right)\right)$$
$$+\alpha\left(1-\frac{h(I^*)}{h(I)}\right)\left(k_3\left(1-\frac{I}{I^*}\right)+\gamma q C^*\left(\frac{C}{C^*}-\frac{I}{I^*}\right)\right).$$

Rewriting the equation, we obtain

$$\dot{L}=-\frac{\mu_1}{S}(S-S^*)^2+S^*\left[\beta C^*+h(I^*)\right]\left(1-\frac{S^*}{S}-\frac{S\left[\beta C+h(I)\right]}{S^*\left[\beta C^*+h(I^*)\right]}+\frac{\left[\beta C+h(I)\right]}{\left[\beta C^*+h(I^*)\right]}\right)$$
$$-\frac{k_2}{CC^*}(C-C^*)^2+S^*\left[\beta C^*+h(I^*)\right]\left(\frac{S\left[\beta C+h(I)\right]}{S^*\left[\beta C^*+h(I^*)\right]}-\frac{C}{C^*}-\frac{SC^*\left[\beta C+h(I)\right]}{S^*C\left[\beta C^*+h(I^*)\right]}+1\right)$$
$$+\alpha k_3\left(1-\frac{h(I^*)}{h(I)}\right)\left(1-\frac{I}{I^*}\right)+\alpha\gamma q C^*\left(1-\frac{h(I^*)}{h(I)}\right)\left(\frac{C}{C^*}-\frac{I}{I^*}\right).$$

Since $\alpha=\frac{S^*h(I^*)}{\gamma q C^*}$, we have

$$\dot{L}=-\frac{\mu_1}{S}(S-S^*)^2-\frac{k_2}{CC^*}(C-C^*)^2+\alpha k_3\left(1-\frac{h(I^*)}{h(I)}\right)\left(1-\frac{I}{I^*}\right)+S^*\beta C^*V_1+S^*h(I^*)V_2$$
$$+S^*h(I^*)\left(\frac{C}{C^*}-\frac{I}{I^*}-\frac{Ch(I^*)}{C^*h(I)}+\frac{Ih(I^*)}{I^*h(I)}\right),$$

where

$$V_1=\left(2-\frac{S^*}{S}-\frac{S}{S^*}\right)\text{ and }V_2=\left(2+\frac{h(I)}{h(I^*)}-\frac{S^*}{S}-\frac{C}{C^*}-\frac{SC^*h(I)}{S^*Ch(I^*)}\right).$$

We observe that $V_1=\left(\frac{2SS^*-S^{*2}-S^2}{SS^*}\right)=\frac{-1}{SS^*}(S-S^*)^2$ and rearranging the last two terms, we have that

$$\dot{L}=-\frac{\mu_1}{S}(S-S^*)^2-\frac{k_2}{CC^*}(C-C^*)^2+\alpha k_3\left(1-\frac{h(I^*)}{h(I)}\right)\left(1-\frac{I}{I^*}\right)-\frac{S^*\beta C^*}{SS^*}(S-S)^2$$
$$+S^*h(I^*)\left(3-\frac{S^*}{S}-\frac{SC^*h(I)}{S^*Ch(I^*)}-\frac{Ch(I^*)}{C^*h(I)}\right)+S^*h(I^*)\left(\frac{h(I^*)}{h(I)}-1\right)\left(\frac{I}{I^*}-\frac{h(I)}{h(I^*)}\right),$$

by conditions II and III, we have

$$\left(1-\frac{h(I^*)}{h(I)}\right)\left(1-\frac{I}{I^*}\right)\leqslant 0,\quad\left(\frac{h(I^*)}{h(I)}-1\right)\left(\frac{I}{I^*}-\frac{h(I)}{h(I^*)}\right)\leqslant 0.$$

Besides $\left(3-\frac{S^*}{S}-\frac{SC^*h(I)}{S^*Ch(I^*)}-\frac{Ch(I^*)}{C^*h(I)}\right)\leqslant 0$. Therefore $\dot{L}\leqslant 0$, for all (S,C,I) and $\dot{L}=0$ if and only if $(S,C,I)=(S^*,C^*,I^*)$. Then the equilibrium is globally asymptotically stable. □

5. Numerical simulations

In this section, we introduce some numerical simulations to show the results previously presented. The model parameters were chosen in order to reflects the COVID-19 dynamics. Following the World Health Organization (WHO), we assumed the global life expectancy, which is 73.4 [30], as the inverse of natural mortality rate of susceptible and recovered individuals. According to the Centers for Disease Control and Prevention (CDC), symptoms may appear 2-14 days after exposure to the virus [5]. The COVID-19 recovery rate was assumed as the inverse of 14 days [2]. Recently, Heuveline (2022) showed that the COVID-19 pandemic reduced the world's life expectancy by about 2 years [13]. Therefore, we assumed that $\mu_2=\mu_3=(71.4)^{-1}$. Once the asymptomatic individuals are not aware of their health status,

it is expected that they have the same influx rate as susceptible individuals ($k_1 = k_2$). On the other hand, less influx of symptomatic infectious individuals is expected ($k_3 < k_1$) due to their health status or quarantine.

To analyze the influence of the function h in the dynamical system, we considered the following functions

$$h(I) = \frac{mI}{a + I}, \tag{5.1}$$

and

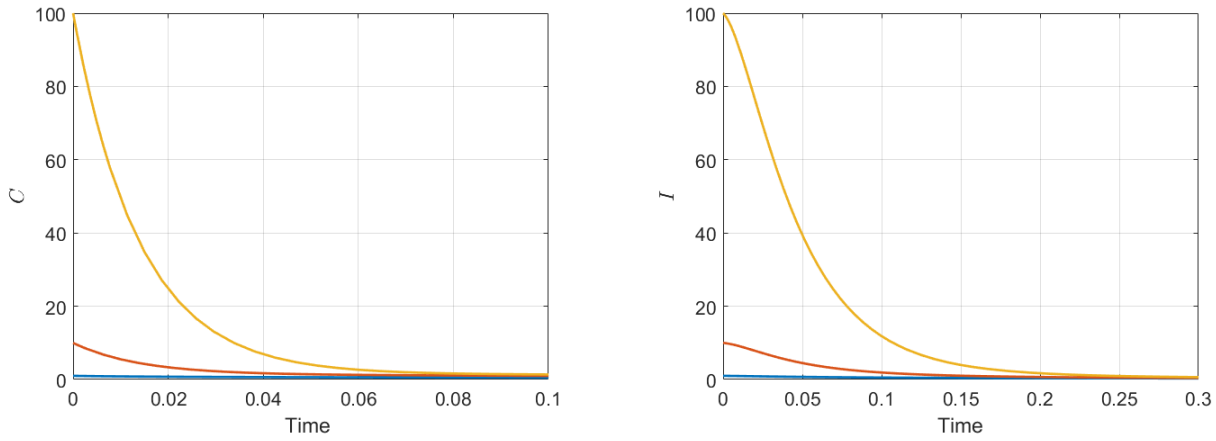
$$h(I) = I^{(1/2)}/(1 + I^{(1/2)}), \tag{5.2}$$

where $m, a > 0$ and h satisfies $h(0) = 0$, $h(I) > 0$, $h'(I) \geqslant 0$, and $h''(I) < 0$. Table 1 shows the model parameters and their description.

Table 1: Parameter description and values adopted in simulations of the system (2.1).

Parameter	Definition	Value	Reference
k_1	Rate of influx of susceptible	0.5	Assumed
β	Transmission rate	0.001	Assumed
μ_1	Death rate of susceptible	1/73.4	[30]
k_2	Rate of influx of carrier compartment	0.5	Assumed
γ	Rate at which carriers develop symptoms	1/(5/365)	[5]
μ_2	Death rate of carriers individuals	1/71.4	[13]
k_3	Rate of influx of infected compartment	0.1	Assumed
q	Fraction of carriers individuals becoming infectious	0.3	[15]
p	Rate of recovery	1/(14/365)	[2]
μ_3	Death rate of infected individuals	1/71.4	[13]
k_4	Rate of influx of recovery compartment	0.4	Assumed
μ_4	Death rate of recovered individuals	1/73.4	[30]
m	Coefficient of function h	0.1	Assumed
a	Coefficient of function h	1	Assumed

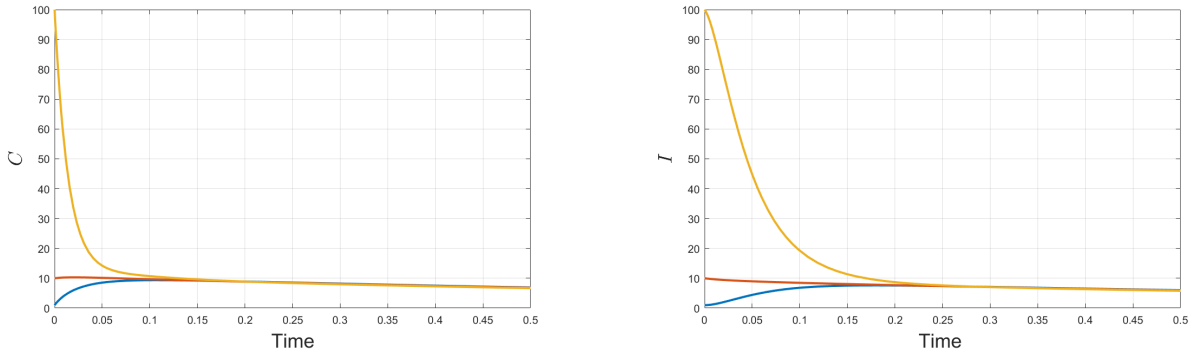
To simulate some epidemiological scenarios, we assumed different initial conditions for infectious and carrier individuals. We assumed the following scenarios $I(0) = C(0) = \theta$, where $\theta = 1, 10, 100$. For the susceptible and recovered individuals we set as 1000 and 0, respectively.



(a) Carrier individuals.

(b) Infected individuals.

Figure 1: Function: $h(I) = mI/(a + I)$. The blue, orange, and yellow colors corresponds to the values of θ: 1, 10, and 100, respectively.



(a) Carrier individuals.

(b) Infected individuals.

Figure 2: Function: $h(I) = I^{(1/2)}/(1 + I^{(1/2)})$. The blue, orange, and yellow colors corresponds to the values of θ: 1, 10, and 100, respectively.

Figure 1 shows the carrier and infected individual dynamics of the system (2.1) with incidence function given by equation (5.1). In Figure 2 we considered a different incidence function, which is given by equation (5.2). In both scenarios, we varied the initial condition and as expected from the globally stability all solutions converge to the respective equilibrium point coordinate.

6. Conclusions

In this work, we analyzed the global stability of the single equilibrium point of a SCIR model with migration terms and nonlinear incidence function. We aimed to analyze the effect of asymptomatic contribution to the infection. The model does not have a basic reproduction number (R_0) since the model has not a free-disease equilibrium point. This is due to the effect of positive constant migration values on the infected and asymptomatic populations. Using Lyapunov's direct method, we proved the global asymptotic stability when considering an incidence of the form $S\,[\beta C + h(I)]$, which is used but we think it may be possible to extend the same results when considering an incidence of the form $f(S)\,[g(C) + h(I)]$. However, the result does not extend immediately, and the question remains open to the scientific community.

To analyze the asymptomatic contribution to the infection, we considered two different incidence functions. From the simulation scenarios we concluded that depending on the incidence function the infection peak can occur more rapidly. Besides, the migration terms contribute to the persistence of the infection.

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References

[1] F. Balloux, L. van Dorp, *Q&A: What are pathogens, and what have they done to and for us?*, BMC biol., **15** (2017), 1–6. 1

[2] H. R. Bhapkar, P. N. Mahalle, N. Dey, K. C. Santosh, *Revisited COVID-19 Mortality and Recovery Rates: Are we Missing Recovery Time Period?*, J. Med. Syst., **44** (2020), 1–5. 5, 1

[3] E. J. Carr, E. J. Carr, J. Dooley, J. E. Garcia-Perez, V. Lagou, J. C. Lee, C. Wouters, I. Meyts, A. Goris, G. Boeckxstaens, M. A. Linterman, A. Liston, *The cellular composition of the human immune system is shaped by age and cohabitation*, Nat. Immunol. **17** (2016), 461–468. 1

[4] F. Castelli, G. Sulis, *Migration and infectious diseases*, Clin. Microbiol. Infect., **23** (2017), 283–289. 1

[5] Centers for Disease Control and Prevention [n.d.], *Symptoms of COVID-19 — CDC*, Accessed 02 September (2022). 5, 1

[6] M. Chen, M. Li, Y. Hao, Z. Liu, L. Hu, L. Wang, *The introduction of population migration to SEIAR for COVID-19 epidemic modeling with an efficient intervention strategy*, Inf. Fusion, **64** (2020), 252–258. 1

[7] M. C. Gómez, E. I. Mondragon, *Global stability analysis for a SEI model with nonlinear incidence rate and asymptomatic infectious state*, Appl. Math. Comput., **402** (2021), 7 pages. 1

[8] M. C. Gómez, E. I. Mondragon, P. L. Molano, *Global stability analysis for a model with carriers and non-linear incidence rate*, J. Biol. Dyn., **14** (2020), 409–420. 1

[9] H. Guo, M. Y. Li, *Impacts of migration and immigration on disease transmission dynamics in heterogeneous populations*, Discrete Contin. Dyn. Syst. Ser. B, **17** (2012), 2413–2430. 1

[10] B. D. Gushulak, D. W. MacPherson, *Globalization of infectious diseases: the impact of migration*, Clin. Infect. Dis., **38** (2004), 1742–1748. 1

[11] W. M. Haddad, V. Chellaboina, Q. Hui, *Nonnegative and compartmental dynamical systems*, Princeton University Press, (2010). 2

[12] J. Hawker, N. Begg, I. Blair, R. Reintjes, J. Weinberg, K. Ekdahl, *Communicable disease control and health protection handbook*, John Wiley & Sons, (2019). 1

[13] P. Heuveline, *Global and National Declines in Life Expectancy: An End-of-2021 Assessment*, Popul. Dev. Rev., **48** (2022), 31–50. 5, 1

[14] G. Huang, C. Nie, Y. Dong, *Global stability for an SEI model of infectious diseases with immigration and age structure in susceptibility*, Int. J. Biomath., **12** (2019), 15 pages. 1

[15] M. A. Johansson, T. M. Quandelacy, S. Kada, P. V. Prasad, M. Steele, J. T. Brooks, R. B. Slayton, M. Biggerstaff, J. C. Butler *SARS-CoV-2 Transmission From People Without COVID-19 Symptoms*, JAMA Netw. Open, **4** (2021), 1–8. 1

[16] B. Kammegne, K. Oshinubi, O. Babasola, O. J. Peter, O. B. Longe, R. B. Ogunrinde, E. O. Titiloye, R. T. Abah, J. Demongeot, *Mathematical Modelling of the Spatial Distribution of a COVID-19 Outbreak with Vaccination Using Diffusion Equation*, Pathogens, **12**, (2023), 1–19. 1

[17] Z. A. Khan, A. L. Alaoui, A. Zeb, M. Tilioua, S. Djilali, *Global dynamics of a SEI epidemic model with immigration and generalized nonlinear incidence functional*, Results Phys., **27** (2021), 1–5. 1

[18] N. Khan, J. Cailhol, *Are migration routes disease transmission routes? Understanding Hepatitis and HIV transmission amongst undocumented Pakistani migrants and asylum seekers in a Parisian suburb*, Anthropol. Med., **27** (2020), 395–411. 1

[19] A. R. Marderstein, M. Uppal, A. Verma, B. Bhinder, Z. Tayyebi, J. Mezey, A. G. Clark, O. Elemento, *Demographic and genetic factors influence the abundance of infiltrating immune cells in human tissues*, Nat. Commun., **11** (2020), 1–14. 1

[20] I. Mokrousov,*Major impact of massive migration on spread of Mycobacterium tuberculosis strains*, Human Migration: Biocultural Perspectives, (2021). 1

[21] M. Naim, F. Lahmidi, A. Namir, *Threshold dynamics of an SEIS epidemic model with nonlinear incidence rates*, Differ. Equ. Dyn. Syst., **2021** (2021), 14 pages. 1

[22] R. Niu, E. W. M. Wong, Y.-C. Chan, M. A. Van Wyk, G. Chen, *Modeling the COVID-19 pandemic using an SEIHR model with human migration*, IEEE Access, **8** (2020), 195503-195514. 1

[23] O. J. Peter, H. S. Panigoro, M. A. Ibrahim, O. M. Otunuga, T. A. Ayoola, A. O. Oladapo, *Analysis and dynamics of measles with control strategies: a mathematical modeling approach*, Int. J. Dyn. Control., (2023), 1–15. 1

[24] R. P. Sigdel, C. C. McCluskey, *Global stability for an SEI model of infectious disease with immigration*, Appl. Math. Comput., **243** (2014), 684–689. 1

[25] S. Y. Tchoumi, H. Rwezaura, M. L. Diagne, G. González-Parra, J. Tchuenche, *Impact of infective immigrants on COVID-19 dynamics*, Math. Comput. Appl., **27** (2022), 1–12. 1

[26] S. Usaini, A. S. Hassan, S. M. Garba, J. S. Lubuma, *Modeling the transmission dynamics of the Middle East Respiratory Syndrome Coronavirus (MERS-CoV) with latent immigrants*, J. Interdiscip. Math., **22** (2019), 903–930. 1

[27] A. Valsamatzi-Panagiotou, R. Penchovsky, *Environmental factors influencing the transmission of the coronavirus 2019: a review*, Environ. Chem. Lett., **20** (2022), 1603–1610. 1

[28] N. Vignier, O. Bouchaud, *Travel, migration and emerging infectious diseases*, EJIFCC, **29** (2018), 175–179. 1

[29] S. Wood, S. E. Harrison, N. Judd, M. A. Bellis, K. Hughes, A. Jones, *The impact of behavioural risk factors on communicable diseases: a systematic review of reviews*, BMC Public Health, **21** (2021), 1–16. 1

[30] World Health Organization, *Life tables*, Accessed 02 September (2022). 5, 1

[31] J. Zhang, J. Li, Z. Ma, *Global dynamics of an SEIR epidemic model with immigration of different compartments*, Acta Math. Sci. Ser. B, **26** (2006), 551–567. 1

[32] R. Zhang, D. Li, S. Liu, *Global analysis of an age-structured SEIR model with immigration of population and nonlinear incidence rate*, J. Appl. Anal. Comput., **9** (2019), 1470–1492. 1