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Global stability analysis of an extended SUC epidemic mathematical model

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Abstract: In this study, we conduct a global stability analysis of an extended Susceptible-Unidentified infected-Confirmed (SUC) epidemic mathematical model. In the original SUC model, the entire population consists of individuals who are susceptible, those with unidentified infections, and those with confirmed infections, without accounting for births and deaths. In the proposed extended SUC model, we incorporate the dynamics of births and deaths into the original SUC model. We analyze the global stability of this extended SUC epidemic mathematical model and perform several computational experiments to validate the global stability analysis. Through this realistic extended SUC model, we aim to advance the current understanding of epidemiological modeling and provide valuable insights for guiding public health interventions and policies.

Keywords: global stability analysis; COVID-19 disease; SUC epidemic model

1 Introduction

Mathematical models are important in understanding and managing COVID-19 because they allow researchers to simulate the spread of the virus [1], predict outcomes under different scenarios, and assess the impact of interventions like social distancing [2], [3], vaccination [3], [4], and lockdowns [5]. These models provide insights into transmission dynamics, estimate the basic reproduction number (R_0) [6], and help allocate healthcare resources efficiently. By predicting potential future outbreaks and guiding public health decisions, mathematical models are essential tools in strategic planning and response to the pandemic and ultimately contribute to reducing the virus's spread and saving lives. The susceptible–infected–removed (SIR) model is the classical model that provides a theoretical framework used to investigate the spreading of the novel COVID-19 disease within a community. In this model, susceptible individuals refer to those who are not currently infected but have the potential to become infected. Infected individuals are those who have already contracted the virus and are capable of transmitting it to susceptible individuals. Removed individuals are those who have either recovered from the virus and are assumed to be immune or have died [7]–[10].

Unlike common epidemic diseases, in the case of the COVID-19 pandemic, infected individuals are typically isolated and do not spread the disease to others, except in rare instances. Therefore, it was necessary to develop a new model specifically targeting the COVID-19 pandemic. Lee et al. [11] proposed a susceptible-unidentified infected-confirmed (SUC) epidemic mathematical equation, which has a structure similar to the SIR model, but with different interpretations for each category. In the next section, we will describe the details of the SUC model and highlight the differences from the SIR model. Using the proposed SUC model and the confirmed case data, Lee et al. [11] could estimate the unidentified infected population. Lee et al. [12] developed a modified SUC model to control the COVID-19 pandemic through financial incentives. Hwang et al. [13] presented a time-dependent SUC model for long-term analysis of the COVID-19 pandemic. In addition, the robust optimal parameters for the SUC epidemic dynamics model were estimated using real-world data [14].

We should note that there are more detailed epidemic models to design more realistic models. Razzaq et al. [15] investigated the behavioral response of population on transmissibility and saturation incidence of a deadly pandemic through a fractional order dynamical system consisting of seven nonlinear fractional order differential equations. Saha et al. [16] have studied the global dynamics of a generalized SIRS epidemic mathematical model that incorporates government policy, public response, and social behavioral reactions. Dutta et al. [17] investigated the dynamics of an epidemic using an SIVIS epidemic mathematical model that accounts for heterogeneous susceptibility, governmental interventions, social behavioral dynamics, and public reactions, considering both autonomous and nonautonomous factors. Another study [18] focused on infectious diseases

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that can be transmitted by asymptomatic carriers who are infected and contagious but do not show any symptoms of the disease. Dutta et al. [19] analyzed a compartmental SIRIS epidemiological mathematical model with two susceptible compartments, considering immunity, government actions, public behavior, and environmental factors. This study emphasized the significance of early governmental intervention, nonlinear dynamics, and sensitivity analysis using Latin hypercube sampling. The findings suggest that a combination of government policies with pharmaceutical therapy significantly reduces disease transmission.

We are well aware that COVID-19 is an infectious disease that has been prevalent for several years. However, the existing SUC model did not include the dynamics of birth and death; therefore, the model is not suitable for analyzing infectious diseases over a long period of time. Therefore, the primary objective of this study is to propose and analyze a modified SUC model that incorporates the dynamics of birth and death.

The remaining parts of this paper are organized as follows. In Section 2, we analyze the global stability of an extended SUC epidemic mathematical model. In Section 3, a numerical solution method is described, and we present a series of computational experiments to validate the global stability analysis. In Section 4, conclusions are drawn.

2 Model formulation and analysis

2.1 Proposed model formulation

We propose an extended Susceptible-Unidentified infected-Confirmed (SUC) epidemic mathematical equation and analyze its global stability analysis of the proposed model. In the original SUC model [11], the entire population N consists of susceptible individuals $S(t)$, individuals with unidentified infections $U(t)$, and those with confirmed infections $C(t)$ at time t:

$$\begin{cases} \dfrac{\mathrm{d}S(t)}{\mathrm{d}t} = -\beta\dfrac{S(t)U(t)}{N}, \\ \dfrac{\mathrm{d}U(t)}{\mathrm{d}t} = \beta\dfrac{S(t)U(t)}{N} - \gamma U(t), \\ \dfrac{\mathrm{d}C(t)}{\mathrm{d}t} = \gamma U(t), \end{cases} \tag{1}$$

where β is the disease transmission rate and γ is the inverse of the days before being confirmed. The SUC model (1) shares structural similarities with the classical susceptible–infected–removed (SIR) epidemic model [8] but has different interpretations of the terms. For a disease such as coronavirus disease 2019 (COVID-19), a highly contagious respiratory illness caused by the SARS-CoV-2 virus that has led to global health crises, once individuals are confirmed to be infected, they are isolate, and no longer transmit the infection. Therefore, the unidentified infected mainly transmit the infection to others. In the initial stages of the COVID-19 outbreak, there was an expectation that it would subside quickly. Contrary to this anticipation, it has persisted for several years. Hence, it is essential to include the dynamics of births and deaths. The original SUC model did not include these dynamics; however, the proposed extended SUC model incorporates them:

$$\begin{cases} \dfrac{\mathrm{d}S(t)}{\mathrm{d}t} = \mu N - \beta\dfrac{S(t)U(t)}{N} - \mu S(t), \\ \dfrac{\mathrm{d}U(t)}{\mathrm{d}t} = \beta\dfrac{S(t)U(t)}{N} - \gamma U(t) - \mu U(t), \\ \dfrac{\mathrm{d}C(t)}{\mathrm{d}t} = \gamma U(t) - \mu C(t). \end{cases} \tag{2}$$

Figure 1 presents schematic diagrams of the different categories in the conventional SIR model and the proposed extended SUC model. The top row of Figure 1 shows the S, I, and R groups in the SIR model. The I group can be further decomposed into two subgroups, UI (unconfirmed-infected population) and CI (confirmed-infected population), see the middle row of Figure 1. In the extended SUC model, U corresponds to UI, and C corresponds to $CI \cup R$, as shown in the bottom row of Figure 1.

In the standard SIR model, γ represents the reciprocal of the duration in which an infected individual develops antibodies and recovers. However, in the proposed extended SUC model, γ denotes the reciprocal of the time span during which an unconfirmed infected individual can transmit the disease before their infection is identified.

We maintain the assumption that $N = S(t) + U(t) + C(t)$ holds true at all times. Therefore, one obtains

$$C(t) = N - S(t) - U(t). \tag{3}$$

Using Eqs. (2) and (3), now it is enough to investigate the following system

$$\begin{cases} \dfrac{\mathrm{d}S(t)}{\mathrm{d}t} = \mu N - \beta\dfrac{S(t)U(t)}{N} - \mu S(t), \\ \dfrac{\mathrm{d}U(t)}{\mathrm{d}t} = \beta\dfrac{S(t)U(t)}{N} - \gamma U(t) - \mu U(t), \end{cases} \tag{4}$$

with the initial data $S(0) > 0$ and $U(0) > 0$.

Figure 1: Schematic diagram of the different categories in the standard SIR model and the proposed extended SUC model.

2.2 Positivity and boundedness of solutions

Define the solution semiflow $\Psi(t)\colon \mathbb{R}^2_+ \to \mathbb{R}^2_+$ of the system (4) by

$$\Psi(t)\psi = (S(t), U(t)), \quad \text{for} \quad t \in \mathbb{R}_+,$$

where $\psi = (S_0, U_0) \in \mathbb{R}^2_+$. Let $\mathcal{D} = \{(S(t), U(t)) \in \mathbb{R}^2_+ : 0 \le S(t) + U(t) \le N\}$. Then, we have the following.

Lemma 2.1. *The set $\mathcal{D}$ is positively invariant for $\Psi(t)$ in the sense of $\Psi(t)\psi \in \mathcal{D}$ for $\psi \in \mathcal{D}$ and $t \ge 0$.*

Proof. From Eq. (4), we get

$$\begin{cases} S(t) = S(0)\exp\left(\int_0^t \left(\frac{\mu N}{S(s)} - \beta\frac{U(s)}{N} - \mu\right)\mathrm{d}s\right), \\ U(t) = U(0)\exp\left(\int_0^t \left(\beta\frac{S(s)}{N} - \gamma - \mu\right)\mathrm{d}s\right). \end{cases}$$

Because we have restricted the initial data $(S(0), U(0)) > (0, 0)$, the solution $(S(t), U(t)) > (0, 0)$ immediately holds for all $t \ge 0$. In the sequel, define $W(t) = S(t) + U(t)$, then one has

$$\frac{\mathrm{d}W(t)}{\mathrm{d}t} = \frac{\mathrm{d}S(t)}{\mathrm{d}t} + \frac{\mathrm{d}U(t)}{\mathrm{d}t} \le \mu N - \mu W(t).$$

It follows that

$$W(t) \le N - \big(N - W(0)\big)\mathrm{e}^{-\mu t},$$

with $W(0) = S(0) + U(0)$. Therefore,

$$\limsup_{t\to+\infty} W(t) = \limsup_{t\to+\infty}(S(t) + U(t)) \le N.$$

The proof follows. □

2.3 Equilibria and stability

Let

$$\begin{cases} F(S, U) = \mu N - \beta\frac{S(t)U(t)}{N} - \mu S(t), \\ G(S, U) = \beta\frac{S(t)U(t)}{N} - \gamma U(t) - \mu U(t). \end{cases} \tag{5}$$

It is clear that system (4) always has a disease-free equilibrium (DFE) $E_0^* = (S_0^*, U_0^*) = (N, 0)$. Now, to perform the stability analysis of DFE, we employ the next-generation approach [20], [21] to first obtain the basic reproduction number of the system (4). To this end, let

$$F = \begin{pmatrix} 0 & 0 \\ 0 & \beta \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} \mu & \beta \\ 0 & \mu + \gamma \end{pmatrix}.$$

As such, the basic reproduction number R_0 of the system (4) can be read as

$$R_0 = \rho(FV^{-1}) = \frac{\beta}{\mu + \gamma}.$$

By direct calculations, the Jacobian matrix of the system (4) at DFE can be given by

$$J^*_{E_0} = \begin{pmatrix} -\frac{\beta U}{N} - \mu & -\frac{\beta S}{N} \\ \frac{\beta U}{N} & \frac{\beta S}{N} - \gamma - \mu \end{pmatrix}_{E_0{}^*} = \begin{pmatrix} -\mu & -\beta \\ 0 & (\mu + \gamma)(R_0 - 1) \end{pmatrix}.$$

Considering $|\lambda E - J_{E_0^*}| = 0$, we can get the following characteristic equation at DFE as below

$$\lambda^2 - T_{E_0^*}\lambda + D_{E_0^*} = 0,$$

where $T_{E_0^*} = (\mu + \gamma)(R_0 - 1) - \mu$ and $D_{E_0^*} = -\mu(\mu + \gamma)(R_0 - 1)$. Therefore, the disease-free equilibrium (DFE) E_0^* is locally asymptotically stable when $R_0 < 1$ and it is unstable when $R_0 > 1$. Let $F(S, U) = G(S, U) = 0$ in Eq. (5), then we know that the system (4) has a unique endemic equilibrium (EE) $E_* = (S_*, U_*)$, where

$$S_* = \frac{(\gamma + \mu)N}{\beta} \quad \text{and} \quad U_* = \frac{\mu N}{\beta}(R_0 - 1).$$

This implies that the endemic equilibrium (EE) $E_* = (S_*, U_*)$ exists only for $R_0 > 1$. Similarly, we can yield the Jacobian matrix of the system (4) at EE as follows

$$J_{E*} = \begin{pmatrix} -\frac{\beta U}{N} - \mu & -\frac{\beta S}{N} \\ \frac{\beta U}{N} & \frac{\beta S}{N} - \gamma - \mu \end{pmatrix}_{E_*} = \begin{pmatrix} -\mu R_0 & -\gamma - \mu \\ \mu(R_0 - 1) & 0 \end{pmatrix}.$$

Considering $|\lambda E - J_{E_*}| = 0$, we can get the following characteristic equation at EE as below

$$\lambda^2 - T_{E_*}\lambda + D_{E_*} = 0,$$

where $T_{E_*} = -\mu R_0$ and $D_{E_*} = \mu(\mu + \gamma)(R_0 - 1)$. We immediately deduce that the endemic equilibrium (EE) $E_* = (S_*, U_*)$ is locally asymptotically stable owing to $R_0 > 1$ holds. We establish the following local stability results of DFE and EE.

Theorem 2.1. *For the SUC epidemic model* (4)*, one claims that*

(i) *The disease-free equilibrium (DFE)* $E_0^* = (N, 0)$ *is locally asymptotically stable as* $R_0 < 1$ *and it is unstable as* $R_0 > 1$;
(ii) *The endemic equilibrium (EE)* $E_* = (S_*, U_*)$ *is locally asymptotically stable as* $R_0 > 1$.

The following result shows the global stability for DFE and EE.

Theorem 2.2. *For the DFE and EE, we have*
(i) *If* $R_0 \le 1$, *then DFE* $E_0^* = (S_0^*, 0)$ *is globally asymptotically stable;*
(ii) *If* $R_0 > 1$ *and* $\mu N \le \sqrt{\mu(\mu N + \beta U_*)S_*}$, *then EE* $E_* = (S_*, U_*)$ *is globally asymptotically stable.*

Proof. For *(i)*, we define

$$V(t) = S(t) - S_0^* - S_0^* \ln \frac{S(t)}{S_0^*} + U(t).$$

Then, we can have

$$\begin{aligned}
\dot{V}(t) &= \frac{\mathrm{d}S(t)}{\mathrm{d}t}\left(1 - \frac{S_0^*}{S(t)}\right) + \frac{\mathrm{d}U(t)}{\mathrm{d}t} \\
&= \left(1 - \frac{S_0^*}{S(t)}\right)\left[\mu N - \beta\frac{S(t)U(t)}{N} - \mu S(t)\right] \\
&\quad + \beta\frac{S(t)U(t)}{N} - \gamma U(t) - \mu U(t) \\
&= \mu\left(N + S_0^* - \frac{S_0^*}{S(t)}N - S(t)\right) + \left(\frac{\beta S_0^*}{N} - \gamma - \mu\right)U(t) \\
&= \mu\left(2N - \frac{N^2}{S(t)} - S(t)\right) + (\gamma + \mu)(R_0 - 1)U(t) \\
&\le (\gamma + \mu)(R_0 - 1)U(t) < 0.
\end{aligned}$$

Hence, the DFE $E_0^* = (S_0^*, 0)$ is globally asymptotically stable.

(ii) Define

$$L(t) = S(t) - S_* - S_* \ln \frac{S(t)}{S_*} + U(t) - U_* - U_* \ln \frac{U(t)}{U_*}.$$

Then, some direct computation shows

$$\begin{aligned}
\dot{L}(t) &= \frac{\mathrm{d}S(t)}{\mathrm{d}t}\left(1 - \frac{S_*}{S(t)}\right) + \frac{\mathrm{d}U(t)}{\mathrm{d}t}\left(1 - \frac{U_*}{U(t)}\right) \\
&= \left(1 - \frac{S_*}{S(t)}\right)\left[\mu N - \beta\frac{S(t)U(t)}{N} - \mu S(t)\right] \\
&\quad + \left(1 - \frac{U_*}{U(t)}\right)\left[\beta\frac{S(t)U(t)}{N} - \gamma U(t) - \mu U(t)\right] \\
&= \mu N - \mu S(t) - (\gamma + \mu)U(t) - \frac{S_*\mu N}{S(t)} + \frac{\beta S_* U(t)}{N} \\
&\quad + \mu S_* - \frac{\beta U_* S(t)}{N} + (\gamma + \mu)U_* \\
&= 2\mu S_* + 2(\gamma + \mu)U_* - \frac{\mu N + \beta U_*}{N}S - \frac{\mu N S_*}{S} \\
&\le 2\mu S_* + 2(\gamma + \mu)U_* - 2\sqrt{\mu(\mu N + \beta U_*)S_*} \\
&= 2\mu N - 2\sqrt{\mu(\mu N + \beta U_*)S_*} < 0,
\end{aligned}$$

where we use $\beta S_*/N = \gamma + \mu$ and $\mu N = (\gamma + \mu)U_* + \mu S_*$. Therefore, the endemic equilibrium EE is globally asymptotically stable. This ends the proof. □

Theorem 2.2 illustrates that the endemic equilibrium EE $E_* = (S_*, U_*)$ is globally asymptotically stable when $R_0 > 1$ and $\mu N \le \sqrt{\mu(\mu N + \beta U_*)S_*}$ are satisfied. The following globally stability result shows that the condition $R_0 > 1$ is enough to ensure the globally stability of the endemic equilibrium EE $E_* = (S_*, U_*)$ by employing a different Lyapunov function.

Theorem 2.3. *For the DFE and EE, we have*
(i) *If* $R_0 \le 1$, *then DFE* $E_0^* = (S_0^*, 0)$ *is globally asymptotically stable;*
(ii) *If* $R_0 > 1$, *then EE* $E_* = (S_*, U_*)$ *is globally asymptotically stable.*

Proof. For *(i)*, we construct the following function

$$\widetilde{V}(t) = \frac{1}{2}\left[(S(t) - S_0^*) + U(t)\right]^2 + \frac{(\gamma + 2\mu)N}{\beta}U(t).$$

Then, the time derivative of $\widetilde{V}(t)$ shows

$$\begin{aligned}
\dot{\widetilde{V}}(t) &= \left[(S(t) - S_0^*) + U(t)\right]\frac{\mathrm{d}(S(t) + U(t))}{\mathrm{d}t} + \frac{(\gamma + 2\mu)N}{\beta}\frac{\mathrm{d}U(t)}{\mathrm{d}t} \\
&= \left[(S(t) - S_0^*) + U(t)\right]\left[\mu N - \mu S(t) - (\gamma + \mu)U(t)\right] \\
&\quad + \frac{(\gamma + 2\mu)N}{\beta}\left[\beta\frac{S(t)U(t)}{N} - (\gamma + \mu)U(t)\right] \\
&= \left[(S(t) - S_0^*) + U(t)\right]\left[-\mu(S(t) - S_0^*) - (\gamma + \mu)U(t)\right] \\
&\quad + \frac{(\gamma + 2\mu)N}{\beta}\left[\beta\frac{S(t)U(t)}{N} - (\gamma + \mu)U(t)\right] \\
&= -\mu(S(t) - S_0^*)^2 - (\gamma + 2\mu)(S(t) - S_0^*)U(t) \\
&\quad - (\gamma + \mu)U^2(t) + \frac{(\gamma + 2\mu)N}{\beta}\left[\beta\frac{S(t)U(t)}{N} - (\gamma + \mu)U(t)\right] \\
&= -\mu(S(t) - S_0^*)^2 - (\gamma + \mu)U^2(t) \\
&\quad + \frac{(\gamma + \mu)(\gamma + 2\mu)S_0^*}{\beta}(R_0 - 1)U(t) \le 0.
\end{aligned}$$

This shows that the DFE $E_0^* = (S_0^*, 0)$ is globally asymptotically stable when $R_0 \le 1$.

(ii) Define

$$\widetilde{L}(t) = \frac{1}{2}[(S(t) - S_*) + (U(t) - U_*)]^2 + \frac{(\gamma + 2\mu)N}{\beta} \times \left[U(t) - U_* - U_* \ln \frac{U(t)}{U_*}\right].$$

By computation, one yields

$$\begin{aligned}
\dot{\widetilde{L}}(t) &= [(S(t) - S_*) + (U(t) - U_*)]\frac{\mathrm{d}(S(t) + U(t))}{\mathrm{d}t} \\
&\quad + \frac{(\gamma + 2\mu)N}{\beta}\frac{\mathrm{d}U(t)}{\mathrm{d}t}\left(\frac{U(t) - U_*}{U(t)}\right) \\
&= [(S(t) - S_*) + (U(t) - U_*)]\big[\mu N - \mu S(t) - (\gamma + \mu)U(t)\big] \\
&\quad + \frac{(\gamma + 2\mu)N}{\beta}\left(\frac{U(t) - U_*}{U(t)}\right)\left[\beta\frac{S(t)U(t)}{N} - (\gamma + \mu)U(t)\right] \\
&= [(S(t) - S_*) + (U(t) - U_*)]\big[-\mu(S(t) - S_*) \\
&\quad - (\gamma + \mu)(U(t) - U_*)\big] + \frac{(\gamma + 2\mu)N}{\beta}(U(t) - U_*) \\
&\quad \times \left[\beta\frac{S(t)}{N} - \beta\frac{S_*}{N}\right] \\
&= -\mu(S(t) - S_*)^2 - (\gamma + \mu)(U(t) - U_*)^2 \le 0,
\end{aligned}$$

where we use $\beta S_*/N = \gamma + \mu$ and $\mu N = (\gamma + \mu)U_* + \mu S_*$. Therefore, one claims that the endemic equilibrium EE is globally asymptotically stable when $R_0 > 1$ holds. This ends the proof. □

3 Numerical solution algorithm and tests

The extended SUC model is numerically solved by using a finite difference method as follows:

$$S_{n+1} = S_n + \Delta t\left(\mu N - \beta\frac{S_n U_n}{N} - \mu S_n\right), \quad n = 0,\ 1,\ 2, \ldots, \tag{6}$$

$$U_{n+1} = U_n + \Delta t\left(\beta\frac{S_n U_n}{N} - \gamma U_n - \mu U_n\right), \tag{7}$$

$$C_{n+1} = N - S_{n+1} - U_{n+1}, \tag{8}$$

where $S_n = S(n\Delta t)$, $U_n = U(n\Delta t)$, and $C_n = C(n\Delta t)$ with a time step Δt. Here, the unknown parameters are β, γ, and U_0. Once these parameter values are determined, we can proceed to solve the discrete system of Eqs. (6)–(8). Due to the condition (3), it is only necessary to solve Eqs. (6) and (7). It is worth noting that a high-order numerical method, such as the fourth-order Runge–Kutta method used in Ref. [22], could also be applied.

Next, we first perform a convergence test of the numerical method to determine an adequately accurate time-step size. Afterward, we conduct various computational experiments

3.1 Convergence test

Before investigating the global stability analysis of the extended SUC epidemic mathematical model, we conduct a convergence test to determine an appropriate temporal step for use in the numerical method. The parameters used are as follows: $N = 10,000$, $C(0) = 700$, $U(0) = 30$, $S(0) = N - U(0) - C(0)$, Days $= 10$, $N_t = \text{Days}/\Delta t$, $\beta = 1.1$, $\gamma = 0.2$, $\mu = 0.05$, and $\mathcal{R}_0 = 4.4$. Table 1 shows the temporal l_2-norm errors and convergence rates with various time steps, where the discrete l_2-norm errors $\|e_{N_t}\|_2$ are defined as follows:

$$\|e_{N_t}\|_2 = \sqrt{\left(S^{\text{ref}} - S_{N_t}\right)^2 + \left(U^{\text{ref}} - U_{N_t}\right)^2 + \left(C^{\text{ref}} - C_{N_t}\right)^2}.$$

Here, S^{ref}, U^{ref}, and C^{ref} are the reference solutions obtained by using a very small time step $\Delta t^{\text{ref}} = 2^{-20}$. The numerical solutions are observed to converge to first-order accuracy. From now on, we will use a time step of $\Delta t = 2^{-11}$.

3.2 Global stability analysis

Let us consider the following parameter values: $N = 10,000, C(0) = 500$, $U(0) = 30, S(0) = N - U(0) - C(0), \beta = 1.1, \gamma = 0.7$, and $\mu = 0.05$. Thus, we obtain the parameters $\mathcal{R}_0 = 4.4$, $S_* = N/\mathcal{R}_0 = 2,272.73$, $U_* = \mu N(\mathcal{R}_0 - 1)/\beta = 1,545.45$, and $C_* = N - S_* - U_* = 6,181.82$. Figure 2 shows the temporal evolution of $S(t), U(t)$, and $C(t)$ until they reach numerical steady states, where the steady states S_s, U_s, and C_s satisfy $\max\{|S_* - S_s|, |U_* - U_s|, |C_* - C_s|\} < 1$ for some smallest integer s.

3.3 Effect of γ on endemic equilibrium values

Next, we investigate the effect of γ on endemic equilibrium values. The parameter γ, which is the inverse of the number of days before being confirmed, affects both the values of the equilibrium state values and the time taken to reach these

Table 1: Temporal l_2-norm errors and convergence rates.

Δt	2^{-8}	Rate	2^{-9}	Rate	2^{-10}	Rate	2^{-11}
$\|e_{N_t}\|_2$	6.4234	1.00	3.2097	1.00	1.6038	1.00	0.8010

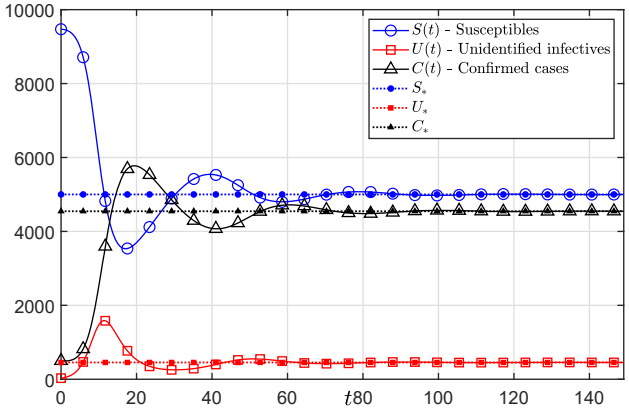

Figure 2: Evolution of $S(t)$, $U(t)$, and $C(t)$ to the endemic equilibrium S_*, U_*, and C_*.

equilibrium states. Here, we use the parameters as follows: $N = 10,000$, $C(0) = 500$, $U(0) = 30$, $\beta = 1.1$, and $\mu = 0.05$. Figure 3(a) shows the values of equilibrium states S_*, U_*, and C_* for the parameter γ and the total population N. Figure 3(b) shows R_0 for the parameter γ. We observe that increasing γ decreases R_0. As the inverse of the days before being confirmed γ increases, unconfirmed infections are identified and isolated more quickly, increasing the equilibrium value of S_*, and decreasing the equilibrium values of C_* and U_*.

Figure 4(a)–(c) show the temporal evolution of the populations $S(t)$, $U(t)$, and $C(t)$ until they reach numerical steady states with $\gamma = 1/2$, $1/4$, and $1/7$, respectively. We can observe that as the value of the parameter γ increases, the number of unidentified infected cases $U(t)$ decreases. However, the time taken to reach the steady state becomes longer. Furthermore, we can observe that when $\gamma = 1/2$, the number of confirmed cases C is smaller compared to when $\gamma = 1/4$ and $\gamma = 1/7$.

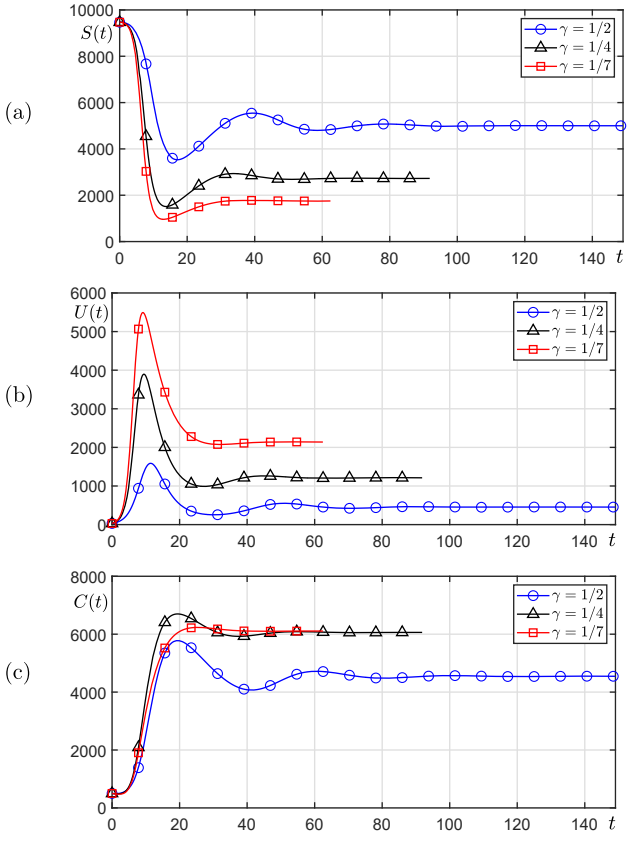

Figure 4: Temporal evolution of populations for (a) $S(t)$, (b) $U(t)$, and (c) $C(t)$ with $\gamma = 1/2, 1/4$, and $1/7$, respectively.

Figure 5 shows the time taken to reach equilibrium states for the parameter γ. We observed that as the value of the parameter γ increases, the time taken to reach numerical steady states becomes longer. However, although it may take more time when γ is large, as we can observe from the results in Figure 4, we have small number of unidentified infected cases.

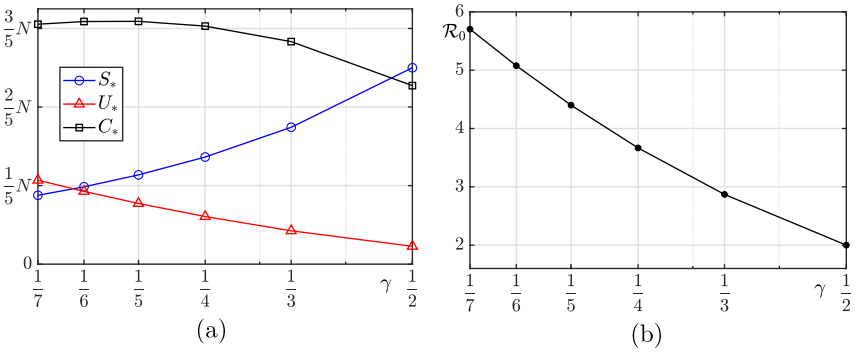

Figure 3: Equilibrium states and basic reproduction numbers. (a) The values of equilibrium states S_*, U_*, and C_* and (b) R_0 for the parameter γ.

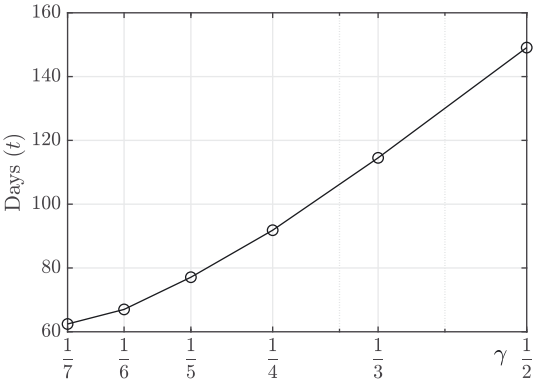

Figure 5: Time taken to reach equilibrium states for the parameter γ.

4 Conclusions

In this study, we have successfully extended the original SUC epidemic model to include the dynamics of births and deaths. The extended novel model offers a more comprehensive representation of population changes over time. Our analysis focused on the global stability of this extended SUC model, and our findings confirm that the system's equilibrium points are globally stable under certain conditions. To validate our theoretical results, we conducted several computational experiments, which consistently supported the global stability analysis. The realistic nature of the extended SUC model provides valuable insights for epidemiological modeling. By incorporating important demographic processes, the model represents real-world situations more closely. Hence, the new model improves its utility in guiding public health interventions and policies. Compared to the SIR model, we can estimate the number of the unidentified infected population from the SUC model and confirmed case data. Scientific community can benefit from the extended SUC model by using it in the development of new epidemic models for predicting epidemic trends and controlling the spread of future pandemic outbreaks.

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