Research Article

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Modelling the adverse impacts of urbanization on human health

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Abstract: Urbanization has been a growing trend for decades, but the Coronavirus disease 2019 (COVID-19) pandemic has accelerated this trend in several ways. In many countries, the pandemic has prompted significant investments in urban health infrastructure, including hospitals, clinics, and public health facilities. Also, there are many studies suggesting that COVID-19, urbanization, and chronic diseases are interconnected in several ways, and addressing one aspect requires a comprehensive approach that considers the interconnectedness of these factors. To date, there are no studies depicting COVID-19 as a cause of emerging chronic diseases within the urban population. To address this sensitive issue, we propose a mathematical model in which we divide the total human population into three compartments: susceptible, diseased, and recovered. We have obtained the equilibrium points of the model and the conditions for their local stabilities. A suitable Lyapunov function has been used to derive the condition for the global stability of the nontrivial equilibrium point. A detailed numerical simulation of the model has been carried out, which is followed by sensitivity analysis of the model system.

Keywords: urbanization, environmental pollution, COVID-19, sensitivity analysis

MSC 2020: 92D30, 34D20

1 Introduction

Urbanization is a process of the mass population shift from rural areas to urban centres, resulting in the growth of cities and the expansion of their boundaries. Coronavirus disease 2019 (COVID-19) has significantly increased urbanization in various ways. It includes the growth of informal settlements due to increased poverty as well as increased suburban development [24]. Moreover, another factor that has contributed to increased urbanization is the rise of e-commerce [1,13]. With more people shopping online, the demand for warehouse and distribution centres has increased, leading to more jobs in urban areas. Additionally, many people have become more reliant on home delivery services during the pandemic, which has further increased the need for these facilities in urban areas. Furthermore, the growing importance of urban centres as hubs of innovation [4,5] and economic activity after COVID-19 is also increasing urban sprawl. With more companies located in cities, and the concentration of talent and resources in these areas, urban centres have become increasingly important for businesses seeking enterprises to remain competitive.

While urbanization can bring about economic growth, social progress, and improved living standards, it also has negative impacts on the environment. One of the most serious implications associated with urbanization is pollution [19–21]. As more people migrate to urban areas, the demand for energy, transportation, goods,
and services increases, leading to higher levels of pollution. This pollution, in turn, has a significant impact on public health [3,15].

In the aforementioned context, it is essential to understand the connection between urbanization and pollution to develop effective strategies for sustainable urban development. Pollution, in all its forms, has become a major concern for human health in recent times. From air pollution to water contamination, the impact of pollution on people’s well-being cannot be ignored.

The issue of pollution has reached such an alarming level that it is now being considered a leading cause of various diseases such as cancer, asthma, heart disease, and neurological disorders. The harmful chemicals and toxins present in the air, water, and soil are making people sick, and the situation is getting worse with each passing day.

The indoor air pollution also emerges as a major threat to the human health. The indoor air pollution received less attention in past but the scenario is changing due to the following two reasons [7]:

1. humans spent, on an average, 80–90% of their life inside the house. In the major part of the globe, houses are poorly ventilated.
2. indoor pollutant levels are typically twice higher compared to the outdoor pollution.

Furthermore, indoor air pollutants (consisting of particulate matter, biological pollutants, physical agents, and over 400 different chemical compounds, mainly volatile organic and inorganic compounds) also cause more than 5 million mortality every year. It also causes multi-millionaire losses due to reduced employee’s productivity, and increased health system expenses [7]. The poor indoor air quality has been classified as a severe challenge to children’s health [9]. A detailed and informative review of indoor air pollutants and its hazardous impacts on human health can be found in [7].

Therefore, it is crucial to understand how pollution is affecting human health and what steps can be taken to mitigate its impact. This article aims to explore the role of urbanization-induced pollution in the deterioration of human health [3,15,27]. Mathematical modelling is an efficient tool for understanding various physical phenomena. In particular, mathematical models are very successful in providing the information about the spread of a particular disease in a certain region [11]. As urbanization can be linked with an increased risk of certain diseases, it can be challenging to determine the precise relationship between urbanization and health outcomes, given the numerous factors that can affect disease development. Mathematical models can help by incorporating data from various sources and simulating the impact of different variables on disease outcomes. For example, a model can be used to analyse the influence of air pollution on respiratory illness in an urban area, integrating data on pollutant levels, population density, and other relevant factors to simulate the risk of respiratory illness under various scenarios. Recently, some new compartmental models pertaining to infectious diseases have been proposed to investigate the impact of environmental pollution on the spread of infectious diseases [14,16,26,29]. In Sharma and Kumari [26] proposed a deterministic model incorporating multiple transmission to investigate the impact of environmental pollution on cholera transmission. The study finds that pollution increases the size of the disease. The mathematical model proposed in [14] investigates the role of pollution in COVID-19. Through a detailed sensitivity analysis, they concluded that pollution plays a positive role in the spread of COVID-19. The study conducted in [29] modified the model proposed in [16] by incorporating environmental noise. Overall, mathematical modelling can offer valuable insights into the complex relationship between urbanization and health outcomes. By modelling various situations, these models can help identify the most crucial factors that contribute to disease risk in urban settings and inform targeted interventions to mitigate those risks. Many studies have been conducted to observe the impacts of environmental changes on the spread of diseases. Some studies had shown the impact of climate change on diseases [2,12,28]. Also, some studies depict that exposure to pollution results in a loss of immunity [8,10,23,25]. The study becomes important because to date no mathematical study has been performed on the role of urbanization in the development of diseases within the human population. Therefore, to address this serious issue, we propose a new compartmental model that considers urbanization as a cause of disease spread within the urban population. The manuscript is divided into seven sections. The mathematical model is given in Section 2. The equilibrium points are obtained in Section 3. The stability analysis of equilibrium points is performed in Section 4. To showcase the impact of urbanization in developing chronic diseases, a detailed numerical simulation is performed in Section 5. Section 6 contains the sensitivity analysis of the model system, which is followed by conclusion and insight into the present study in Section 7.
2 Mathematical model

Based on the literature survey on urbanization and its ill impacts, the following points can be highlighted:
(a) COVID-19 played a significant role in growth of urbanization, which, in turn, is polluting the environment.
(b) Regular exposure to pollution is a leading cause of several chronic diseases.

Hence, we propose a compartmental model to study the impact of urbanization on the depreciation of human health. We assume that a total human population \( N \) is divided into three mutually exclusive compartments \( H \), \( E \), and \( R \). Here, \( H \) represents the human population susceptible to diseases caused by urbanization, \( E \) represents the diseased population, and \( R \) represents the recovered individuals. It is assumed that the susceptible population gets the disease due to regular exposure to pollution. Other crucial assumptions of the model are highlighted as follows:
(1) Let \( m \) represent the density of urbanization programmes and \( P \) be the pollution caused by them.
(2) We further assume that
\[
\frac{dm}{dt} \propto H \quad \frac{dP}{dt} \propto m \quad \frac{dE}{dt} \propto HP.
\]
(3) Let \( A \) be the natural growth rate of the human population \( H \), and \( \beta \) be the rate by which the population is getting the disease. Individuals from class \( R \) enter into class \( H \) with a rate of \( \theta_0 \).
(4) Natural mortality rate of all individuals is \( d \), disease-related death rate is \( \mu \), while \( \theta \) represents the recovery rate of the diseased population.
(5) \( a \) represents the rate of increase in the density of urbanization programmes due to an increase in human population \( H \), and \( a_0 \) is the rate of failure of these programmes.
(6) We say that \( \gamma \) represents the rate of increase of environmental pollution, while \( \gamma_0 \) is the natural decay rate of pollution.

All the parameters involved in the model are positive and initial conditions satisfy the following inequalities:
\[
m \geq 0, \quad P \geq 0, \quad H \geq 0, \quad E \geq 0, \quad R \geq 0.
\]

Using all these assumptions, the proposed deterministic nonlinear model is formulated as follows:
\[
\frac{dm}{dt} = aH - a_0m, \tag{1}
\]
\[
\frac{dP}{dt} = \gamma m - \gamma_0 P, \tag{2}
\]
\[
\frac{dH}{dt} = A - \beta HP - dH + \theta_0 R, \tag{3}
\]
\[
\frac{dE}{dt} = \beta HP - \theta E - dE - \mu E, \tag{4}
\]
\[
\frac{dR}{dt} = \theta E - dR - \theta_0 R. \tag{5}
\]

Figure 1 depicts the schematic diagram of the proposed model system. To perform the study, we first obtain the region of attraction \( \Omega \) as follows:
\[
\Omega = \left\{ 0 \leq H, E, R \leq \frac{A}{d}, \quad 0 \leq m \leq m_q, \quad 0 \leq P \leq P_q \right\},
\]
where \( m_q = \frac{aA}{a_0d} \) and \( P_q = \frac{\gamma A}{\gamma_0 d} \).
In this section, we investigate the existence of equilibrium points for the proposed mathematical model. The model system possess two equilibrium points:

(i) trivial equilibrium point \( E_1 = (0, 0, H^0, 0, 0) \), where \( H^0 = \frac{A}{a} \).

(ii) nontrivial equilibrium point.

The solution of the following set of equations gives the nontrivial equilibrium point as \( E_2 = (m^*, P^*, H^*, E^*, R^*) \) for the proposed model system.

\[
\begin{align*}
\alpha H^* - \alpha_0 m^* &= 0, \\
\gamma m^* - \gamma_0 P^* &= 0, \\
A - \beta H^* P^* - dH^* + \theta_0 R^* &= 0, \\
\beta H^* P^* - \theta E^* - dE^* - \mu E^* &= 0, \\
\theta E^* - dR^* - \theta_0 R^* &= 0.
\end{align*}
\]

From equation, we obtain (6)

\[ m^* = \frac{aH^*}{a_0}, \]

and from equation (7),

\[ P^* = \frac{ym^*}{\gamma_0} = \frac{ayH^*}{a_0\gamma_0}. \]

Next, equation (9) gives

\[ E^* = \frac{\beta H^* P^*}{\theta + d + \mu} = \frac{\beta ayH^2}{(\theta + d + \mu)a_0\gamma_0}. \]

From equation (10),

\[ R^* = \frac{\theta E^*}{d + \theta_0} = \frac{\theta_0 ayH^2}{(d + \theta_0)(\theta + d + \mu)a_0\gamma_0}. \]

Substituting the value of \( P^* \) and \( R^* \) into equation (8), we obtain a quadratic equation in \( H^* \) as,

\[ F(H^*) = a_1 H^2 + a_2 H^* + a_3 = 0, \]
where

\[ a_1 = \beta \alpha \gamma (d \theta + \theta_0(d + \mu)), \]
\[ a_2 = a_0(d + \theta_0)(\theta + d + \mu), \]
\[ a_3 = -A a_0 \gamma d + \theta_0)(\theta + d + \mu). \]

It is evident from the expressions of \( a_1 \), \( a_2 \), and \( a_3 \) that \( a_3 \) is always negative. By Descart's rule of sign, equation (11) always has a unique positive root.

## 4 Stability analysis

In this section, we discuss the stability of the equilibrium points obtained in Section 3. First, we study the stability of trivial equilibrium points \( E_1 \) followed by the stability of the nontrivial equilibrium point \( E_2 \).

### 4.1 Stability of trivial equilibrium point

The Jacobian matrix of the model system (1)–(5) around the trivial equilibrium point is given by

\[
\mathcal{F} = \begin{bmatrix}
-a_0 & 0 & a & 0 & 0 \\
0 & -\gamma_0 & 0 & 0 & 0 \\
0 & \frac{-\beta A}{d} & -d & 0 & \theta_0 \\
0 & \frac{\beta A}{d} & 0 & -\theta - d - \mu & 0 \\
0 & 0 & 0 & \theta & -d - \theta_0 \\
\end{bmatrix}
\]

The characteristic polynomial of \( \mathcal{F} \) is

\[
A_1 \lambda^5 + A_2 \lambda^4 + A_3 \lambda^3 + A_4 \lambda^2 + A_5 \lambda + A_6 = 0,
\]

(12)

where

\[
A_1 = d, \\
A_2 = d(a_0 + 3d + \theta_0 + \theta + \mu + \gamma_0), \\
A_3 = a_0 d(3d + \theta_0 + \theta + \mu + \gamma_0) + d(d + \theta_0)(\theta + 2d + \mu) + d(\theta + d + \mu)(d + \gamma_0) + d\gamma_0(2d + \theta_0), \\
A_4 = a_0 d(\theta + d + \mu)(2d + \theta_0 + \gamma_0) + \gamma_0 d(d + \theta_0)(a_0 + \theta + 2d + \mu) + a_0 d^2 (d + \theta_0 + \gamma_0) \\
+ d^2 (\theta + d + \mu)(d + \theta_0 + \gamma_0) + \beta \alpha \gamma a, \\
A_5 = da_0 d(\theta + \gamma_0)(d + \theta_0)(d + \theta + \mu) + d\gamma_0(d + \theta_0)(a_0 + d + \theta_0) + \beta A \alpha a(\theta + 2d + \mu + \theta_0), \\
A_6 = a_0 \gamma_0 d^2 (\theta + d + \mu)(d + \theta_0) + \beta \gamma a(A \theta + d + \mu)(d + \theta_0) - \theta d \alpha \gamma A).
\]

By the Routh-Hurwitz criterion, the system is locally stable if and only if all the coefficients of the characteristic polynomial are positive and they satisfy the following inequalities:

\[
B_1 = \frac{A_2 A_3 - A_1 A_4}{A_2} > 0, \quad C_1 = \frac{A_4 (A_2 A_3 - A_1 A_4) - A_2 (A_2 A_5 - A_1 A_6)}{A_2 A_3 - A_1 A_4} > 0, \quad \text{and} \quad \frac{A_2 A_5 - A_1 A_6}{A_2} - \frac{B_1 A_6}{C_1} > 0.
\]

### 4.2 Stability of nontrivial equilibrium point

The Jacobian matrix of model systems (1)–(5) around \( E_2 \) is given by
The characteristic polynomial of \( V \) is
\[
K_6\lambda^5 + K_5\lambda^4 + K_4\lambda^3 + K_3\lambda^2 + K_2\lambda + K_1 = 0,
\]
where
\[
K_1 = 1,
K_2 = (a_0 + 3d + \theta_0 + \theta + \mu + \gamma_0 + \beta P^*),
K_3 = \beta P^*(\theta + 2d + \mu + \theta_0 + \gamma_0 + a_0) + d[2(\theta + \theta_0 + \mu) + 3(d + a_0 + \gamma_0)].
\]
By the Routh-Hurwitz criterion, the system is locally stable if and only if all the coefficients of the characteristic polynomial are positive and they satisfy the following inequalities:
\[
R_1 = \frac{K_2K_4 - K_1K_6}{K_2} > 0, \quad S_1 = \frac{K_4(K_2K_4 - K_1K_6) - K_6(K_2K_6 - K_1K_4)}{K_2K_3 - K_1K_4} > 0, \quad \text{and} \quad \frac{K_2K_5 - K_1K_6}{K_2} - \frac{R_1K_6}{S_1} > 0.
\]

### 4.3 Global stability of nontrivial equilibrium point

In this section, we derive the condition of global stability of the nontrivial equilibrium point \( E_2 \). To achieve this goal, we use an appropriate Lyapunov function and obtain the following theorem.

**Theorem 1.** The nontrivial equilibrium point \( E_2 \) is globally stable under the following condition:
\[
\frac{2\theta_0^2\theta^2}{\beta P^*(\theta + d + \mu)^2(\theta + d + \mu)} < \min \left\{ \frac{4\gamma_0d^2(\theta + d + \mu)^2}{27(\beta A^2)}, \frac{4\gamma_0^2d^2(\theta + d + \mu)^2P^*}{y^2A^2} \right\}.
\]

To establish the global stability of the nontrivial equilibrium point \( E_2 \), we consider the following Lyapunov function:
\[
V = \frac{C_1}{2}(H - H^*)^2 + \frac{C_2}{2}(E - E^*)^2 + \frac{C_3}{2}(R - R^*)^2 + \frac{C_4}{2}(m - m^*)^2 + \frac{C_5}{2}(P - P^*)^2,
\]
where the unknowns \( C_i, \ i = 1, 2, 3, 4, 5 \) need to be chosen appropriately. On differentiating \( V \) along the solution of the proposed model system, we obtain
\[
\frac{dV}{dt} = -C_1\beta P^*(H - H^*)^2 - C_2d(H - H^*)^2 - C_3\theta d + \mu(E - E^*)^2 - C_3(\theta + d + \mu)(R - R^*)^2 - C_4a_0(m - m^*)^2 - C_5d(\theta + d + \mu)(R - R^*)^2 - C_6\gamma_0d(\theta + d + \mu)(R - R^*)^2 - C_7\beta H(P - P^*)(H - H^*) + C_8d(P - P^*)(R - R^*)(H - H^*) + C_9\beta E(E - E^*)(H - H^*) + C_9\beta P^*(E - E^*)(H - H^*) + C_9\theta(E - E^*)(R - R^*) + C_9a(m - m^*)(H - H^*) + C_9\gamma(m - m^*)(P - P^*).
\]
Now, \( \frac{dV}{dt} \) will be negative definite under the following inequalities:
Now, we choose $C_1 = 1$, $C_2 = \frac{2a_{0}}{\beta P^{*}(d + a_{0})}$, $C_3 = \frac{a_{0} P^{*}}{\alpha}$, $C_4 = \frac{3\beta P^{*}}{\beta P^{*}(d + \mu)} < C_5 < \frac{a_{0} P^{*}(d + \mu)}{3P^{*}}$ and a positive value of $C_2$ in such a way that condition stated in the theorem satisfied.

5 Numerical simulation

Numerical simulation is a crucial tool to solve complex model equations and obtain meaningful results from them. Using it, we can make predictions and conclusions out of our model system.

Since the real-world data are not available for study, most of the parametric values given below are chosen from the literature [6,16,17,22]:

$$A = 100, \beta = 0.018, d = 0.035, \theta = 0.012, \mu = 0.01, \theta_0 = 0.085, \alpha = 0.2, y = 0.15, a_0 = 0.1125, \text{ and } y_0 = 0.094$$

The main objective of this research is to investigate the impact of COVID-19-induced urbanization on the growth of chronic illness among the human population. In order to achieve this goal, parameters such as $\alpha$, $a_0$, $y$, $y_0$ and $\beta$ are crucial as it is necessary to observe how changes in these parameters affect the variation of the diseased population ($E$).

In Figure 2(a), we have studied the variation of the diseased population ($E$) with time for different values of $\alpha$. The figure shows that as the rate of increase in the density of urbanization programmes ($\alpha$) increases, the number of diseased individuals also escalates. Similarly, from Figure 2(b), it can be concluded that the diseased population ($E$) increases with an increase in the rate of environmental pollution ($y$).

Moreover, Figure 2(c) demonstrates the variation of the diseased population ($E$) with time for different values of $a_0$. The results indicate that a larger value of $a_0$ and a smaller value of $\alpha$ can significantly reduce the incidence of diseases ($E$) compared to a higher value of $\alpha$ has a more significant impact in increasing the number of individuals affected by diseases ($E$) compared to $y$ (Figure 2(a) and (b)).

The plot represented in Figure 4(a) depicts the influence of $a_0$ on the diseased population ($E$). The results indicate that a larger value of $a_0$ and a smaller value of $\alpha$ can significantly reduce the incidence of diseases
Figure 2: Time series plot of diseased population ($E$) for varying values of $\alpha$, $\gamma$, $\alpha_0$, and $\gamma_0$: (a) time series plot of diseased population ($E$) for varying values of $\alpha$, (b) time series plot of diseased population ($E$) for varying values of $\gamma$, (c) time series plot of diseased population ($E$) for varying values of $\alpha_0$, and (d) time series plot of diseased population ($E$) for varying values of $\gamma_0$.

Figure 3: Variation of diseased population ($E$) with $\alpha$ and $\gamma$: (a) variation of diseased population with $\alpha$ for varying values of $\gamma$ and (b) variation of diseased population with $\gamma$ for varying values of $\alpha$. 

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[Graphs and plots are included here.]
among individuals. Conversely, as $\alpha_0$ decreases and approaches zero, the entire population becomes increasingly susceptible to contracting illnesses.

Similarly, the impact of $\gamma_0$ on the diseased population ($E$) is studied in Figure 4(b), which reveals similar trends. As the value of $\gamma_0$ increases, the number of individuals affected by illnesses ($E$) reduces significantly. However, as $\gamma_0$ approaches zero, the incidence of diseases among individuals increases substantially.

The analysis of Figure 5 reveals the impact of $\beta$ and $\alpha$ on the diseased population ($E$). It is observed that when $\beta$ is small, the variation in $\alpha$ has a minimal effect on the number of diseased individuals. However, as $\beta$ increases, the prevalence of illnesses also rises significantly.

Similarly, Figure 6 demonstrates the impact of $\beta$ and $\gamma$ on the diseased population ($E$). The figure highlights that the number of individuals afflicted with illnesses ($E$) increases with a rise in $\beta$, and an increase in $\gamma$ exacerbates the situation further. Consequently, it can be inferred that a rise in environmental pollution is likely to lead to a surge in the prevalence of diseases among individuals.

Figure 7 sheds light on the influence of $\beta$ and $\alpha_0$ on the prevalence of diseased individuals. The analysis of the figure reveals that an escalation in the value of $\beta$ leads to a proportional increase in the diseased population ($E$). Conversely, an increase in the value of $\alpha_0$ has an inverse relationship with the prevalence of illnesses, as an increase in $\alpha_0$ results in a decline in the number of diseased individuals. Therefore, it can be inferred that when urbanization programmes fail more frequently, the rate of disease occurrence among individuals diminishes.

Figure 4: Variation of diseased population ($E$) with $\alpha_0$ and $\gamma_0$: (a) variation of diseased population with $\alpha_0$ for varying values of $\alpha$ and (b) variation of diseased population with $\gamma_0$ for varying values of $\alpha$.

Figure 5: Surface plot showing simultaneous impact of $\beta$ and $\alpha$ on the diseased population.
Following a thorough analysis of Figure 8, it can be inferred that the increase in $\beta$ is directly correlated with a rise in the number of diseased individuals. Conversely, augmenting the rate of natural decay of pollution $\gamma_0$ leads to a significant decrease in the diseased population ($E$). Taking into consideration Figures 5–8, it is evident that the fluctuation in the density of urbanization programmes and environmental pollution
is progressively resulting in a larger number of people becoming afflicted with illnesses. However, it can also be concluded that a reduction in pollution levels and urban sprawl is proving to be highly effective in limiting the prevalence of diseased individuals.

To establish the stability of the $E_2$, we plotted the trajectories of the diseased population ($E$) in relation to the population of individuals who are susceptible ($H$) in Figure 9(a), and the diseased population ($E$) in relation to the recovered individuals ($R$) in Figure 9(b). Notably, all trajectories converge toward $E_2$, indicating that the point is stable in both the $H - E$ and $R - E$ phase planes.

Figure 10 shows the time-series plot of the diseased population with unrestricted and minimal urban sprawl. We also see from the figure that if efforts are made in the direction of sustainable development, the number of diseased individuals can be controlled to a greater extent.

6 Sensitivity analysis

Sensitivity analysis is a useful technique for understanding how a model system behaves when its parameters are varied. In this section, we will conduct a comprehensive sensitivity analysis to investigate the impact of
several key parameters, including $\alpha$, $\gamma$, $\alpha_0$, and $\gamma_0$. To do this, we will calculate the sensitivity of the system with respect to each of these parameters as follows:

\[
\begin{align*}
\dot{m}(t, a) &= aH(t, a) + H(t, a) - a\dot{m}(t, a), \\
\dot{P}(t, a) &= \gamma m(t, a) - \gamma_0 P(t, a), \\
\dot{H}(t, a) &= -\beta [H(t, a)P(t, a) + P(t, a)H(t, a)] - dH(t, a) + \theta \dot{R}(t, a), \\
\dot{E}(t, a) &= \beta [H(t, a)P(t, a) + P(t, a)H(t, a)] - \theta E(t, a) - dE(t, a) - \mu E(t, a), \\
\dot{R}(t, a) &= \theta E(t, a) - dR(t, a) - \theta \dot{R}(t, a), \\
\end{align*}
\]

\[
\begin{align*}
\dot{m}(t, y) &= aH(t, y) - a_0m(t, y), \\
\dot{P}(t, y) &= \gamma m(t, y) + m(t, y) - \gamma_0 P(t, y), \\
\dot{H}(t, y) &= -\beta [H(t, y)P(t, y) + P(t, y)H(t, y)] - dH(t, y) + \theta \dot{R}(t, y), \\
\dot{E}(t, y) &= \beta [H(t, y)P(t, y) + P(t, y)H(t, y)] - \theta E(t, y) - dE(t, y) - \mu E(t, y), \\
\dot{R}(t, y) &= \theta E(t, y) - dR(t, y) - \theta \dot{R}(t, y), \\
\end{align*}
\]

\[
\begin{align*}
\dot{m}(t, a_0) &= aH(t, a_0) - a_0m(t, a_0) - m(t, a_0), \\
\dot{P}(t, a_0) &= \gamma M(t, a_0) - \gamma P(t, a_0), \\
\dot{H}(t, a_0) &= -\beta [H(t, a_0)P(t, a_0) + P(t, a_0)H(t, a_0)] - dH(t, a_0) + \theta \dot{R}(t, a_0), \\
\dot{E}(t, a_0) &= \beta [H(t, a_0)P(t, a_0) + P(t, a_0)H(t, a_0)] - \theta E(t, a_0) - dE(t, a_0) - \mu E(t, a_0), \\
\dot{R}(t, a_0) &= \theta E(t, a_0) - dR(t, a_0) - \theta \dot{R}(t, a_0), \\
\end{align*}
\]

Figure 11: Semi-relative sensitivity plots for state variables (a) $H$, (b) $E$ and (c) $R$ with respect to the parameters $\alpha$, $\gamma$, $\alpha_0$, and $\gamma_0$. 

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and
\[
\begin{align*}
\dot{m}(t, y_0) &= \alpha H(t, y_0) - \alpha_0 m_0(t, y_0), \\
\dot{P}(t, y_0) &= \gamma m_0(t, y_0) - P(t, y_0) - \gamma_0 P_0(t, y_0), \\
\dot{H}(t, y_0) &= -\beta H(t, y_0) P(t, y_0) + P_0(t, y_0) H(t, y_0) - dH(t, y_0) + \theta_0 R(t, y_0), \\
\dot{E}(t, y_0) &= \beta H(t, y_0) P(t, y_0) + P_0(t, y_0) H(t, y_0) - \theta E(t, y_0) - dE(t, y_0) - \mu E(t, y_0), \\
\dot{R}(t, y_0) &= \theta E(t, y_0) - dR(t, y_0) - \theta_0 R_0(t, y_0),
\end{align*}
\]
respectively.

Figure 11 displays the semi-relative solutions of the three state variables concerning the parameters \(\alpha\), \(\gamma\), \(\alpha_0\), and \(\gamma_0\). The plots provide valuable information on how the state variables change when a particular parameter is doubled in value.

It shows that when \(\alpha\) is doubled, the susceptible population decreases by 18, while the diseased population escalates by 200. Also, doubling \(\alpha\) increases the recovered individuals by 20.

On the same line, doubling the value of \(\gamma\) is decreasing the susceptible population by 11 and increasing the diseased population and recovered population by 157 and 17, respectively.

Next, on doubling \(\alpha_0\), the diseased population and recovered population are decreasing by 139 and 17, respectively. However, it escalates the value of the susceptible population by 17. Finally, when \(\gamma_0\) is doubled, it has a negative impact of 102 and 10 on the diseased population and recovered population, respectively. However, it can be concluded that it increases the susceptible population by 9.

![Figure 12](image-url)

**Figure 12:** Logarithmic sensitivity plots for the state variables (a) \(H\), (b) \(E\), and (c) \(R\) with respect to the parameters \(\alpha\), \(\gamma\), \(\alpha_0\), and \(\gamma_0\).
In summary, the graphs in Figure 11 provide valuable information on how the state variables change in response to variations in the parameters. The results suggest that changes in $\alpha$, $\gamma$, $\alpha_0$, and $\gamma_0$ have different impacts on the state variables and they can play a pivotal role in deciding the dynamics of disease development.

Figure 12 depicts the logarithmic sensitivity solutions for three state variables $H$, $E$, and $R$ in relation to the parameters $\alpha$, $\gamma$, $\alpha_0$, and $\gamma_0$. These plotted curves represent the percentage change in solutions that occur when a particular parameter is doubled. Essentially, the graph shows the impact of doubling a parameter on the resulting solution, expressed as a percentage change.

It can be observed that on doubling the parameter $\alpha$, the diseased population and recovered population increase by 69 and 29%, respectively. On the other hand, the susceptible population decreases by 20%.

Moreover, doubling the value of $\gamma$ results in an increment of 55 and 22% in diseased population and recovered population, respectively. Furthermore, the susceptible population also decreases by 12% when the value of $\gamma$ is doubled.

Next, on doubling the value of $\alpha_0$, the diseased population and recovered population decrease by 48 and 22%, respectively. It is noteworthy that the susceptible population increases by 19% when the value of $\alpha_0$ is doubled. Furthermore, doubling $\gamma_0$ decreases the diseased population and recovered population by 36 and 13%, respectively. Moreover, doubling the value of $\gamma_0$ also escalates the value of the susceptible population by 10%.

7 Conclusion

In this article, we proposed a compartmental model to understand the role of urbanization in the development of chronic diseases within the human population post COVID-19. We discussed in Section 1 how COVID-19 had induced urban sprawl, which, in turn, is degrading human health in long run. Considering this serious issue, we proposed a mathematical model in which we divided the total human population into three categories, namely, susceptible, diseased, and recovered. Numerical analysis of the model supported the fact that urban sprawl is playing a crucial role in increasing the number of diseased individuals in human society.

Furthermore, sensitivity analysis of the model brought forth the fact that urbanization and pollution are largely affecting the susceptible, diseased, and recovered population. The variation of the diseased population ($E$) with respect to the parameters $\alpha$ and $\gamma$ (Figure 3) shows a rise in the value of $E$ as the value of these parameters increases. However, the increment in diseased population is much sharper when the value of $\beta$ is also increased along with $\alpha$ and $\gamma$ (Figures 5 and 6). Figure 3 with the time-series plots of the diseased population (Figure 2) also revealed the fact that increasing $\alpha$ (which is the rate of increase in density of urbanization programmes) and $\gamma$ (rate of increase of environmental pollution) is drastically impacting the number of diseased individuals. Similarly, we can conclude from Figure 4 that when $\alpha_0$ (rate of failure of urbanization programmes) and $\gamma_0$ (natural decay rate of pollution) are increased, the trajectories of the diseased population ($E$) start declining.

The sensitivity analysis of the model system showed that $\alpha$, $\gamma$, $\alpha_0$, and $\gamma_0$ are greatly affecting the number of individuals in different classes of our model system.

The insights of this study can be summarized as follows:
(a) Urbanization (induced by COVID-19) plays a significant role in developing chronic diseases within the human population.
(b) The rate of increase in the density of urbanization programmes ($\alpha$) and the rate of increase in environmental pollution ($\gamma$) are playing a pivotal role in developing diseases in urban settlements.
(c) An increase in the rate of failure of urbanization programmes ($\alpha_0$) and the natural decay rate of pollution ($\gamma_0$) can help diminish the number of diseased individuals.
(d) To the best of our knowledge, the present study is the only mathematical effort made in this direction and the findings of this study are consistent with the ecological study conducted in [18].

In conclusion, the COVID-19 pandemic has led to significant changes in urbanization patterns around the world. This shift in urbanization has highlighted the need for more flexible and resilient urban planning...
strategies. As cities continue to grow and change in response to the pandemic, it is crucial to prioritize the needs of all residents, including those who are most vulnerable. Overall, the COVID-induced shift in urbanization patterns presents both challenges and opportunities for urban planners and policymakers as unrestricted urban sprawl may lead to a rise in chronic diseases due to several environmental factors. As urbanization continues to spread, it is important for governments, health professionals, and individuals to take steps to mitigate the risks associated with chronic diseases. This can be achieved by creating more green spaces and promoting urbanization in a controlled manner. Through these efforts, we can create healthier urban environments that promote well-being and prevent chronic diseases.

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**References**


