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Research Article

Investigating Respiratory Disease Transmission Patterns Around the Figuil Cement Works

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Abstract

The objective of this research is to examine the dissemination of respiratory illnesses, exacerbated by the airborne pollutants emitted by the Figuil cement works, among the local population residing in the vicinity. The primary objective is to examine the impact of pollution, particularly the emission of fine particles and noxious gases, on the transmission of respiratory diseases such as asthma, chronic bronchitis and other lung disorders. The modified SEIR (Susceptible-Exposed-Infected-Recovered) epidemiological model is employed for the analysis of transmission dynamics within the community. This model incorporates environmental, health and demographic variables, thereby enabling the simulation of disease transmission as a function of varying pollution levels. Particular emphasis is placed on vulnerable groups, such as children and the elderly, due to immunosenescence, who are more likely to suffer from the adverse effects of pollution. The results will facilitate the formulation of efficacious strategies, including the implementation of awareness-raising campaigns and the introduction of sophisticated systems for the filtration and capture of pollutants at their source, such as fine particle filters or devices for the reduction of nitrogen oxides (NOx), with the objective of limiting the spread of respiratory diseases in at-risk areas and the formulation of suitable control measures.

Keywords

Air Pollution, Respiratory Diseases, Atmospheric Pollution, Figuil Cement Plant, Fine Particles, Modified SEIR Epidemiological Model

1. Introduction

In Cameroon, there is a paucity of empirical research examining the impact of cement and marble works on respiratory health. However, the available data suggest a correlation between exposure to emissions from cement works and an increase in respiratory diseases in the surrounding areas. The Figuil Cement and Marble Works, situated in the northern region of Cameroon, represents a significant source of employment and economic development for the region. However, the industrial activities of this cement plant, in particular the production of cement and marble, result in the genera-

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tion of considerable emissions of atmospheric pollutants, notably fine particles (PM2.5 and PM10), sulphur oxides (SO_2), and nitrogen oxides (NOx) [1, 2]. These pollutants, which are known to have adverse effects on human health, are of particular concern to the local population residing in the vicinity of the cement plant.

The issue of air pollution in this region has given rise to growing concerns about its impact on public health, particularly in relation to the spread of respiratory diseases. The prevalence of respiratory diseases, including asthma, chronic bronchitis and lung infections, is exacerbated by continuous exposure to elevated levels of air pollutants. Such illnesses can disseminate rapidly throughout populations, particularly in regions where access to healthcare is constrained. A substantial body of research has demonstrated a clear correlation between exposure to air pollution and an elevated prevalence of respiratory illnesses. For example, the World Health Organisation (WHO) has highlighted that fine particles are particularly capable of penetrating deep into the lungs, causing inflammation and exacerbating conditions such as asthma and chronic obstructive pulmonary disease (COPD) [1, 4, 6, 8]. In the Figuil locality, studies on air quality are limited, but local reports and testimonies from residents indicate an increased prevalence of respiratory diseases, particularly in children and the elderly, who are more vulnerable to the effects of pollution [1]. The SEIR (Susceptible-Exposed -Infected-Recovered) epidemiological model is a widely used tool for understanding the spread of infectious diseases within populations. Extensions of this model have been employed to examine the dynamics of respiratory diseases in settings where pollution exerts a catalytic influence. Recent research has incorporated environmental factors, such as air quality, into epidemiological models with the objective of improving understanding of the influence of air pollutants on the spread of respiratory diseases [6, 10]. These models simulate the impact of fluctuations in pollution on infection and recovery rates. A number of studies have demonstrated that populations residing in close proximity to industrial areas are at an elevated risk of adverse health outcomes due to the sustained inhalation of toxic pollutants. Children, in particular, are more susceptible to developing chronic respiratory diseases due to their still-developing immune systems [3, 9]. Conversely, the elderly, due to immunosenescence, are more likely to develop respiratory infections and encounter serious complications. To improve air quality and reduce the incidence of respiratory diseases, targeted interventions are required. These include the adoption of cleaner industrial technologies, the introduction of advanced systems for filtering and capturing pollutants at source to reassure air quality monitoring, and raising awareness among local populations of the dangers of pollution. Prevention models incorporating epidemiological and environmental data can be used to develop more effective public health strategies to limit the spread of respiratory diseases in high-risk areas such as Figuil.

2. Patterns of Respiratory Disease Transmission Around the Figuil Cement and Marble Works

The prevalence of respiratory diseases in the vicinity of the Figuil cement and marble works can be attributed to a multitude of factors associated with industrial pollution. The activities of the cement and marble works result in the generation of fine particles and other airborne pollutants, which have an adverse impact on the respiratory health of the surrounding population. Modelling the respiratory diseases associated with air pollution around the Figuil Cement and Marble Works is of paramount importance for the comprehension and mitigation of the health impacts of this source of pollution. In the conventional approach to modelling, a number of age compartments are included in order to reflect the differing susceptibility and responses to pollutants exhibited by different age groups. Nevertheless, a streamlined methodology can offer a comprehensive overview while remaining effective for management and prevention objectives. In order to streamline the analysis while capturing the essential dynamics of respiratory disease transmission, we propose reducing the initial model [5, 7, 8] to two main compartments: children and the elderly. Children are particularly vulnerable due to their developing immune systems and potentially increased exposure to pollutants. Their susceptibility to developing chronic respiratory diseases justifies their separation into a separate compartment. The elderly, due to the natural deterioration of the immune system and lung function, are also very sensitive to the effects of pollution. Including them in a separate compartment makes it easier to understand and address the specific needs of this population.

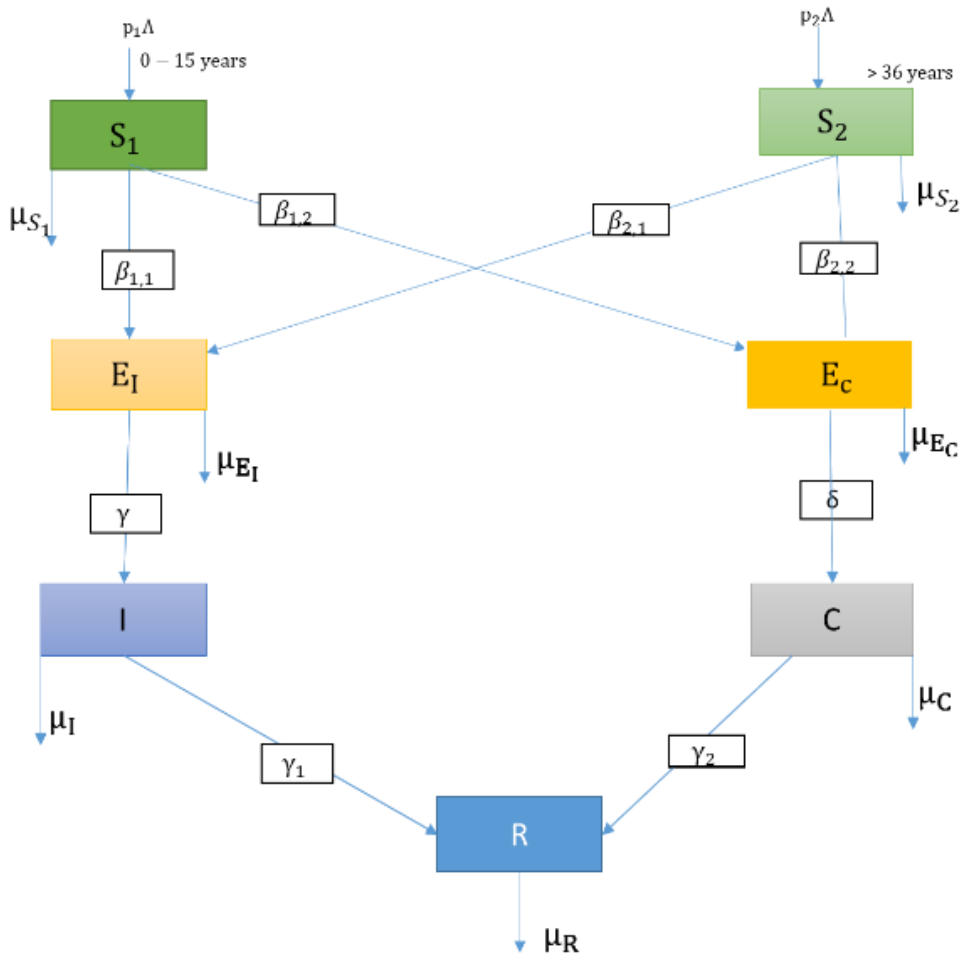

Figure 1. *The following compartmental diagram illustrates the transmission model of respiratory disease in the vicinity of the Figuil cement and marble works.*

The diagram presents a simplified epidemiological model that elucidates the transmission of respiratory disease among the local population in the vicinity of the Figuil cement and marble works. The model captures the dynamics of respiratory disease by considering different transmission scenarios within the population, with a particular focus on critical ages. The model does not account for vertical transmission and is composed of two age classes: individuals aged 0 to 15 (class S_1) and individuals aged over 36 (class S_2). The transition between the different compartments is governed by the rates described below.

The terms S_1 and S_2 are used to represent the compartments of susceptible individuals, that is to say, those who are healthy but who may potentially become infected. The term S_1 refers to the age group between 0 and 15 years old, while S_2 refers to individuals aged over 36. The presence of air pollution has been demonstrated to increase the susceptibility of individuals to disease, as well as the severity of infection. This is reflected by its influence on the transmission and progression rates ($p_i\Lambda$) in each S_i class. The two groups may become exposed to the disease via the transmission rates $\beta_{1,1}$, $\beta_{1,2}$, $\beta_{2,1}$ and $\beta_{2,2}$, which determine the probability of a susceptible individual becoming exposed after contact with an infectious person.

The compartments E_I and E_C represent those of exposed individuals who are not yet infectious. An individual's transition from the susceptible state to the exposed state is contingent upon their interaction with infectious individuals and their transmission rate β. Similarly, the transition from S_1 to E_I and from S_2 to E_C is also dependent upon the level of exposure to the contaminant (polluted air).

I represents the compartment of individuals who have been infected and are capable of transmitting the disease. Individuals in compartment E_I can evolve towards compartment I at a rate determined by γ, which marks the beginning of the period during which they can infect other individuals.

The letter C represents the compartment of individuals who have developed a chronic form of the disease. It is possible that some individuals, particularly those in the E_C compartment, may progress to a chronic state at a rate δ. These individuals are no longer infectious, but their state of health deteriorates over the long term.

R represents the compartment of individuals who have recovered from the disease. Recovery may occur from the

infectious compartment (I) at a rate designated as γ_1, or from the chronic compartment (C) at a rate designated as γ_2.

The model parameters are as follows:

$p_i\Lambda$ represents the proportion of the population exposed to pollution (contaminated air) from the cement plant recruited in each class S_i.

The transmission rates of the disease between the different age groups, $\beta_{1,1}$, $\beta_{1,2}$, $\beta_{2,1}$ and $\beta_{2,2}$, are represented by these variables.

The natural or disease-related mortality rate of individuals in class i is represented by μ_i.

The rates of progression of exposure to the chronic and infectious states are represented by δ and γ.

The rates of recovery of infected individuals and individuals in the chronic phase to the recovered compartment R are represented by γ_1 and γ_2.

The differential equations that govern the movement of individuals between the different health states (susceptible, exposed, infectious, chronic, recovered) in the epidemiological model for the transmission of respiratory diseases around the Figuil cement plant are given by equation (1).

$$\begin{cases} \dot{S}_1 = p_1\Lambda - \mu_{S_1}S_1 - (\beta_{1,1}I + \beta_{1,2}I)S_1 \\ \dot{S}_2 = p_2\Lambda - \mu_{S_2}S_2 - (\beta_{2,1}I + \beta_{2,2}I)S_2 \\ \dot{E}_I = (\beta_{1,1}I + \beta_{1,2}I)S_1 - (\mu_{E_I} + \gamma)E_I \\ \dot{E}_C = (\beta_{2,1}I + \beta_{2,2}I)S_2 - (\mu_{E_C} + \delta)E_C \\ \dot{I} = \gamma E_I - (\mu_I + \gamma_1)I \\ \dot{C} = \delta E_C - (\mu_C + \gamma_2)C \\ \dot{R} = \gamma_1 I + \gamma_2 C - \mu_R R \end{cases} \quad (1)$$

2.1. Positivity of Solutions

In order to demonstrate that our epidemiological model permits positive and bounded solutions, we will analyse the differential equations of the system and examine their behaviour over time. In particular, we aim to show that the populations in each compartment $(S_1, S_2, E_I, E_C, I, C, R)$ remain positive and do not exceed certain limits. This implies that the populations in each compartment never become negative. To verify this, we will analyse each equation.

Compartiment S_1(or S_2):

The equation of S_1 is:

$$\dot{S}_1 = p_1\Lambda - \mu_{S_1}S_1 - (\beta_{1,1}I + \beta_{1,2}I)S_1$$

It is assumed that the initial condition, $S_1(0)$, is positive, that is to say, $S_1(0) \geq 0$. This implies that the population in compartment S_1 is positive at the outset.

The term $p_1\Lambda$ represents a positive constant, denoting the inflow into compartment S_1. The terms $\mu_{S_1}S_1$ and $(\beta_{1,1}I + \beta_{1,2}I)S_1$are negative terms that serve to reduce the population of S_1.

In the event that S_1 reaches zero, the equation $\dot{S}_1 = p_1\Lambda > 0$ indicates that S_1 cannot remain at zero and will begin to increase due to the positive inflow $p_1\Lambda$. It can therefore be concluded that $S_1(t)$ will remain positive.

If S_1 is positive, the differential equation $\dot{S}_1 = p_1\Lambda - \mu_{S_1}S_1 - (\beta_{1,1}I + \beta_{1,2}I)S_1$ is a form of the Riccati-type relation, which often admits bounded solutions under certain conditions. It is necessary to ensure that $S_1(t)$ remains positive; this can be achieved by verifying that the loss rate $(\mu_{S_1} + \beta_{1,1}I + \beta_{1,2}I)S_1$ is never greater than the input $p_1\Lambda$. In other words, the objective is to demonstrate that $p_1\Lambda$ is greater than or equal to $(\mu_{S_1} + \beta_{1,1}I + \beta_{1,2}I)S_1(t)$. This is true as long as $S_1(t)$ is sufficiently small, that is to say, $S_1(t) \leq \frac{p_1\Lambda}{(\mu_{S_1}+\beta_{1,1}I+\beta_{1,2}I)}$.

Therefore, when $S_1(t)$ is positive but small, $S_1(t)$ remains positive as long as the terms $(\mu_{S_1} + \beta_{1,1}I + \beta_{1,2}I)$ do not exceed $p_1\Lambda$. It can be similarly reasoned that $S_2(t)$ also remains positive.

Compartment E_I (or E_C):

For the sake of argument, we may assume that E_I can become negative, that is to say, $E_I < 0$. Substituting this into the equation $\dot{E}_I = (\beta_{1,1}I + \beta_{1,2}I)S_1 - (\mu_{E_I} + \gamma)E_I$, we obtain: Substituting $E_I < 0$ into the equation yields: The temporal derivative of E_I is given by the following equation: $\dot{E}_I = (\beta_{1,1}I + \beta_{1,2}I)S_1 - (\mu_{E_I} + \gamma)E_I$ The variable E_I is therefore defined as:

Given that E_I is less than zero and that $\mu_{E_I} + \gamma$ are positive (representing the death rate and progression rate), the term $\mu_{E_I} + \gamma$ Thus, E_I is positive. It can be seen that the right-hand side of the equation is: The equation can be rearranged as follows: $(\beta_{1,1}I + \beta_{1,2}I)S_1 - (\mu_{E_I} + \gamma)E_I$ It can be demonstrated that E_I will be greater than: $(\beta_{1,1}I + \beta_{1,2}I)S_1$, given that the value of $-(\mu_{E_I} + \gamma)E_I$ is less than zero. The value of E_I is positive. Consequently, the term $-(\mu_{E_I} + \gamma)E_I$ will exert a positive influence on $\dot{E}_I$. If E_I were initially negative, the temporal derivative (E_I) would be positive, indicating an increase in E_I towards positive values. This is inconsistent with the assumption that E_I could remain negative. Therefore, it can be concluded that E_I must be positive or zero. It can therefore be concluded that, by applying a similar line of reasoning to that used for E_I, E_C must remain positive or zero.

Compartment I (or C):

For the sake of argument, let us assume that I can become negative, that is to say, I<0. This leads to the equation $\dot{I} = \gamma E_I - (\mu_I + \gamma_1)I$.

Given that I is less than zero and the term $(\mu_I + \gamma_1)I$ is positive, it follows that the latter will add a positive term to γE. Therefore, we can conclude that $\dot{I}$ will be positive. If the derivative of I with respect to time, $\dot{I}$ is positive while I is less than zero, it follows that I will increase towards positive values. This is in contradiction with the initial assumption that I can remain negative. It follows that I must be positive

or zero. The same line of reasoning can be applied to C, which also guarantees the positivity of the solutions.

Compartment R:

It is assumed that R can become negative, that is to say, $R < 0$. Given that $\dot{R} = \gamma_1 I + \gamma_2 C - \mu_R R$, the term $\gamma_1 I + \gamma_2 C$ is positive. If R is negative, then the term $-\mu_R R$ is positive, given that $-\mu_R R$ is positive and R is negative. Therefore, when R is less than zero, the term $-\mu_R R$ will be positive and increase the rate of change of R (i.e. $\dot{R}$). If R were negative, then $\dot{R}$ would be positive. This implies that R will increase towards positive values due to the term $-\mu_R R$ being positive. Consequently, R cannot remain negative; it must be positive or zero.

2.2. The Originality of the Solutions

The objective of this demonstration is to prove that the solutions are bounded. This is to be achieved by establishing the existence of positive constants M_{S_1}, M_{S_2}, M_{E_I}, M_{E_C}, M_I, M_C, M_R such that: It is also required that $S_1(t) \leq M_{S_1}, S_2(t) \leq M_{S_2}, E_I(t) \leq M_{E_I}, E_C(t) \leq M_{E_C}, I(t) \leq M_I, C(t) \leq M_C, R(t) \leq M_R$.

The total sum N(t) of individuals in all compartments at a given time t is given by the following equation:

$$N(t) = S_1(t) + S_2(t) + E_I(t) + E_C(t) + I(t) + C(t) + R(t)$$

The total derivative can now be calculated.

$$\frac{dN(t)}{dt} = \dot{S_1}(t) + \dot{S_2}(t) + \dot{E_I}(t) + \dot{E_C}(t) + \dot{I}(t) + \dot{C}(t) + \dot{R}(t)$$

$$\Leftrightarrow \frac{dN(t)}{dt} = [p_1\Lambda - \mu_{S_1}S_1 - (\beta_{1,1}I + \beta_{1,2}I)S_1 + p_2\Lambda - \mu_{S_2}S_2 - (\beta_{2,1}I + \beta_{2,2}I)S_2 + (\beta_{1,1}I + \beta_{1,2}I)S_1 - (\mu_{E_I} + \gamma)E_I + (\beta_{2,1}I + \beta_{2,2}I)S_2 - (\mu_{E_C} + \delta)E_C + \gamma E_I - (\mu_I + \gamma_1)I + \delta E_C - (\mu_C + \gamma_2)C + \gamma_1 I + \gamma_2 C - \mu_R R]$$

In essence, the positive and negative terms associated with the transmission of the infection are in equilibrium, resulting in the following equation:

$$\Leftrightarrow \frac{dN(t)}{dt} = p_1\Lambda + p_2\Lambda - (\mu_{S_1}S_1 + \mu_{S_2}S_2 + \mu_{E_I}E_I + \mu_{E_C}E_C + \mu_I I + \mu_C C + \mu_R R)$$

Adjustment for natural variation in mortality:

$\frac{dN(t)}{dt} = \Lambda(p_1 + p_2) - \mu N(t)$ in this equation, μ represents a weighted average of natural mortality rates. The general solution to this linear differential equation is as follows:

$$N(t) = \frac{\Lambda(p_1+p_2)}{\mu} + \left(N(0) - \frac{\Lambda(p_1+p_2)}{\mu}\right)e^{-\mu t}$$

This shows that N(t) is bounded by $\frac{\Lambda(p_1+p_2)}{\mu}$.

It can be demonstrated that a positive constant, M_N, exists such that N(t) is bounded by M_N for all $t \geq 0$. This is due to the fact that N(t) is the sum of the compartments $S_1(t), S_2(t), E_I(t)$, and therefore the result follows from the boundedness of these individual compartments. Given that $E_C(t), I(t), C(t), R(t)$ and N(t) are bounded, it follows that each compartment $S_1(t), S_2(t), E_I(t), E_C(t), I(t), C(t), R(t)$ is also bounded. It follows that there exist positive constants M_{S_1}, M_{S_2}, M_{E_I}, M_{E_C}, M_I, M_C, M_R such that: This implies that the total population size cannot grow indefinitely and is therefore bounded.

The inequalities are as follows:

$$S_1(t) \leq M_{S_1}, S_2(t) \leq M_{S_2}, E_I(t) \leq M_{E_I}, E_C(t) \leq M_{E_C}, I(t) \leq M_I, C(t) \leq M_C, R(t) \leq M_R.$$

2.3. The Existence and Uniqueness of Solutions

The given system of differential equations can be expressed as a Cauchy problem.

$$\dot{X}(t) = F(X(t)) \text{ with } X(t) = \begin{pmatrix} S_1(t) \\ S_2(t) \\ E_I(t) \\ E_C(t) \\ I(t) \\ C(t) \\ R(t) \end{pmatrix} \text{ and}$$

$$F(X(t)) = \begin{pmatrix} p_1\Lambda - \mu_{S_1}S_1 - (\beta_{1,1}I + \beta_{1,2}I)S_1 \\ p_2\Lambda - \mu_{S_2}S_2 - (\beta_{2,1}I + \beta_{2,2}I)S_2 \\ (\beta_{1,1}I + \beta_{1,2}I)S_1 - (\mu_{E_I} + \gamma)E_I \\ (\beta_{2,1}I + \beta_{2,2}I)S_2 - (\mu_{E_C} + \delta)E_C \\ \gamma E_I - (\mu_I + \gamma_1)I \\ \delta E_C - (\mu_C + \gamma_2)C \\ \gamma_1 I + \gamma_2 C - \mu_R R \end{pmatrix}$$

The Cauchy problem is then formulated as follows: The initial value X(0) is equal to X_0, where X_0 represents the given initial conditions. The function F(X(t)) is infinitely differentiable on $\mathbb{R}_+^7$, and thus locally Lipschitzian there. The Cauchy-Lipschitz theorem allows us to conclude that there exists a unique maximum solution to the Cauchy problem associated with the differential equation (1) for the initial condition $(t_0, X_0) \in \mathbb{R}_+^7$. Furthermore, since F(X(t)) is of class C^∞, this solution is also of class C^∞.

2.4. Basic Reproduction Number (R_0)

The calculation of the infection-free equilibrium point (DFE) is as follows: the DFE is reached when $E_I = E_C = I = C = 0$. Assuming that the populations of susceptible S_i are at equilibrium, we can write:: $\begin{cases} 0 = p_1\Lambda - \mu_{S_1}S_1 \\ 0 = p_2\Lambda - \mu_{S_2}S_2 \end{cases}$

The solutions are as follows: $(S_1^*, S_2^*, E_I, E_C, I, C, R) =$

$$\left(\frac{p_1\Lambda}{\mu_{S_1}}, \frac{p_2\Lambda}{\mu_{S_2}}, 0, 0, 0, 0, 0\right)$$

The basic reproduction number (R_0) is calculated using the spectrum of the Jacobian matrix evaluated at the DFE. This matrix is formed by linearising the system around the DFE. The new infection rate matrix (F) and the transition rate matrix between infected compartments (V) are given by:

$$F = \begin{bmatrix} (\beta_{1,1}I + \beta_{1,2}I)S_1^* \\ (\beta_{2,1}I + \beta_{2,2}I)S_2^* \\ 0 \\ 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} (\mu_{E_I} + \gamma)E_I \\ (\mu_{E_C} + \delta)E_C \\ -\gamma E_I + (\mu_I + \gamma_1)I \\ -\delta E_C + (\mu_C + \gamma_2)C \end{bmatrix}$$

The Jacobians of F and V are evaluated in the absence of infection, whereby $E_I = E_C = I = C = 0$.

$$\mathbf{F} = \frac{\partial \mathbf{F}}{\partial(E_I, E_C, I, C)} = \begin{pmatrix} \beta_{1,1}S_1^* & 0 & \beta_{1,2}S_1^* & 0 \\ 0 & \beta_{2,1}S_2^* & 0 & \beta_{2,2}S_2^* \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

$$\mathbf{V} = \frac{\partial \mathbf{V}}{\partial(E_I, E_C, I, C)} = \begin{pmatrix} \mu_{E_I} + \gamma & 0 & 0 & 0 \\ 0 & \mu_{E_C} + \delta & 0 & 0 \\ -\gamma & 0 & \mu_I + \gamma_1 & 0 \\ 0 & -\delta & 0 & \mu_C + \gamma_2 \end{pmatrix}$$

The basic reproduction number, denoted by R_0, is given by two distinct formulas: $R_0 = \rho(-FV^{-1})$ with $\rho(A)$. In the first formula, ρ is the spectral radius of the matrix $-FV^{-1}$, which is defined as the dominant eigenvalue. This formula is applicable when $S_P(A)$ represents the spectrum of A. In the second formula, A is the matrix whose eigenvalues are the solutions of the characteristic equation:

$\det(-FV^{-1} - \lambda I) = 0.$

$$V^{-1} = \begin{pmatrix} \frac{1}{\mu_{E_I} + \gamma} & 0 & 0 & 0 \\ 0 & \frac{1}{\mu_{E_C} + \delta} & 0 & 0 \\ \frac{\gamma}{(\mu_{E_I} + \gamma)(\mu_I + \gamma_1)} & 0 & \frac{1}{\mu_I + \gamma_1} & 0 \\ 0 & \frac{\delta}{(\mu_{E_C} + \delta)(\mu_C + \gamma_2)} & 0 & \frac{1}{\mu_C + \gamma_2} \end{pmatrix}$$

The matrix FV^{-1} is given by the equation

$$FV^{-1} = \begin{pmatrix} \frac{\beta_{1,1}S_1^*}{\mu_{E_I}+\gamma} + \frac{\beta_{1,2}S_1^*\gamma}{(\mu_{E_I}+\gamma)(\mu_I+\gamma_1)} & 0 & \frac{\beta_{1,2}S_1^*}{\mu_I+\gamma_1} & 0 \\ 0 & \frac{\beta_{2,1}S_2^*}{\mu_{E_C}+\delta} + \frac{\beta_{2,2}S_2^*\delta}{(\mu_{E_C}+\delta)(\mu_C+\gamma_2)} & 0 & \frac{\beta_{2,2}S_2^*}{\mu_C+\gamma_2} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

$$\det(-FV^{-1} - \lambda I) = \lambda^2 \left(\frac{\beta_{1,1}S_1^*}{\mu_{E_I}+\gamma} + \frac{\beta_{1,2}S_1^*\gamma}{(\mu_{E_I}+\gamma)(\mu_I+\gamma_1)} - \lambda\right)\left(\frac{\beta_{2,1}S_2^*}{\mu_{E_C}+\delta} + \frac{\beta_{2,2}S_2^*\delta}{(\mu_{E_C}+\delta)(\mu_C+\gamma_2)} - \lambda\right)$$

The expression for R_0 can be approximated by the following approximation:

$$R_0 \approx \max\left\{\frac{\beta_{1,1}S_1^*}{\mu_{E_I}+\gamma} + \frac{\beta_{1,2}S_1^*\gamma}{(\mu_{E_I}+\gamma)(\mu_I+\gamma_1)}, \quad \frac{\beta_{2,1}S_2^*}{\mu_{E_C}+\delta} + \frac{\beta_{2,2}S_2^*\delta}{(\mu_{E_C}+\delta)(\mu_C+\gamma_2)}\right\}$$

2.5. The Overall Stability of the Disease-Free Equilibrium Point (DFE) Is of Significant Interest in This Context

Theorem 1: In the case of system (1), if $R_0 \leq 1$, then the DFE is globally asymptotically stable along the positive orthant $\mathbb{R}_+^7$. Conversely, if $R_0 > 1$, the DFE is unstable.

Proof: In order to study the stability of the DFE, it is necessary to analyse the eigenvalues of the Jacobian matrix of the system evaluated at the DFE [8]. If all the eigenvalues have a negative real part, the DFE is locally asymptotically stable. It will be demonstrated that the initial situation arises when $R_0 \leq 1$. The Jacobian matrix of the system (1) evaluated at the disease-free equilibrium is given by J(0) = F + V. Given that F is non-negative and V is a stable Metzler matrix, it follows that F + V is a regular decomposition of J(0). Therefore, by virtue of [5], we can conclude that $\rho(-FV^{-1})$ is equivalent to where α(M) denotes the stability modulus of the matrix M, which is defined as the largest real part of the elements of its spectrum. It follows that the disease-free equilibrium is locally asymptotically stable. This, in turn, implies, in accordance with Hirsch's theorem, that the disease-free equilibrium (in this case, the origin) is globally asymptotically stable if $R_0 = \rho(-FV^{-1}) < 1$.

2.6. The Existence of an Endemic Equilibrium

Proposition: In the case of system (1), a unique endemic equilibrium point $(S_1^*, S_2^*, E_I^*, E_C^*, I^*, C^*, R^*)$ is obtained by

solving system (1) with, for example, the substitution method in the positive orthant, provided that $R_0 > 1$.

Theorem 2: (The overall stability of the endemic balance is of paramount importance.) In the event that R_0 is greater than 1, the single endemic equilibrium point $(S_1^*, S_2^*, E_I^*, E_C^*, I^*, C^*, R^*)$ of system (1) is globally asymptotically stable.

Proof: In order to examine the system's overall stability, we will simplify the model equation by grouping the infectious latents E_I and chronic latents E_C into a single class labelled I_1, and the infectious (I) and chronic patients (C) into a second class labelled I_2. The resulting simplified model is as follows:

$$\begin{cases} \dot{S}_1 = p_1\Lambda - \mu_{S_1}S_1 - \left(\beta_{1,1}I_1 + \beta_{1,2}I_2\right)S_1 \\ \dot{S}_2 = p_2\Lambda - \mu_{S_2}S_2 - \left(\beta_{2,1}I_1 + \beta_{2,2}I_2\right)S_2 \\ \dot{I}_1 = \left(\beta_{1,1}I_1 + \beta_{1,2}I_2\right)S_1 + \left(\beta_{2,1}I_1 + \beta_{2,2}I_2\right)S_2 - \left(\mu_{E_I} + \gamma + \mu_{E_C} + \delta\right)I_1 \\ \dot{I}_2 = (\delta + \gamma)I_1 - (\mu_I + \gamma_1 + \mu_C + \gamma_2)I_2 \\ \dot{R} = (\gamma_1 + \gamma_2)I_2 - \mu_R R \end{cases} \tag{2}$$

The distinctive endemic equilibrium point $(S_1^*, S_2^*, I_1^*, I_2^*, R^*)$ is defined by the following relationships:

$$\begin{cases} 0 = p_1\Lambda - \mu_{S_1}S_1^* - \left(\beta_{1,1}I_1^* + \beta_{1,2}I_2^*\right)S_1^* \\ 0 = p_2\Lambda - \mu_{S_2}S_2^* - \left(\beta_{2,1}I_1^* + \beta_{2,2}I_2^*\right)S_2^* \\ 0 = \left(\beta_{1,1}I_1^* + \beta_{1,2}I_2^*\right)S_1^* + \left(\beta_{2,1}I_1^* + \beta_{2,2}I_2^*\right)S_2^* - \left(\mu_{E_I} + \gamma + \mu_{E_C} + \delta\right)I_1^* \\ 0 = (\delta + \gamma)I_1^* - (\mu_I + \gamma_1 + \mu_C + \gamma_2)I_2^* \\ 0 = (\gamma_1 + \gamma_2)I_2^* - \mu_R R^* \end{cases} \tag{3}$$

The following Lyapunov candidate function is worthy of consideration:

$$V = (S_1 - S_1^* \ln S_1) + (S_2 - S_2^* \ln S_2) + (I_1 - I_1^* \ln I_1) + \frac{\beta_{1,2}S_1^* + \beta_{2,2}S_2^*}{\mu_I + \gamma_1 + \mu_C + \gamma_2}(I_2 - I_2^* \ln I_2)$$

The derivative of the Lyapunov candidate function V along trajectories of the ordinary differential system (2) is given by the following expression:

$$\dot{V} = \left(p_1\Lambda - \mu_{S_1}S_1 - (\beta_{1,1}I_1 + \beta_{1,2}I_2)S_1 - p_1\Lambda\frac{S_1^*}{S_1} + \mu_{S_1}S_1^* + S_1^*(\beta_{1,1}I_1 + \beta_{1,2}I_2)\right) + \left(p_2\Lambda - \mu_{S_2}S_2 - (\beta_{2,1}I_1 + \beta_{2,2}I_2)S_2 - p_2\Lambda\frac{S_2^*}{S_2} + \mu_{S_2}S_2^* + S_2^*(\beta_{2,1}I_1 + \beta_{2,2}I_2)\right) + \left((\beta_{1,1}I_1 + \beta_{1,2}I_2)S_1 + (\beta_{2,1}I_1 + \beta_{2,2}I_2)S_2 - (\mu_{E_I} + \gamma + \mu_{E_C} + \delta)I_1 - I_1^*\left(\beta_{1,1} + \beta_{1,2}\frac{I_2}{I_1}\right)S_1 + I_1^*\left(\beta_{2,1} + \beta_{2,2}\frac{I_2}{I_1}\right)S_2 - (\mu_{E_I} + \gamma + \mu_{E_C} + \delta)I_1^*\right) + \frac{\beta_{1,2}S_1^* + \beta_{2,2}S_2^*}{\mu_I + \gamma_1 + \mu_C + \gamma_2}\left((\delta + \gamma)I_1 - (\mu_I + \gamma_1 + \mu_C + \gamma_2)I_2 - I_2^*(\delta + \gamma)\frac{I_1}{I_2} - (\mu_I + \gamma_1 + \mu_C + \gamma_2)I_2^*\right)$$

By employing the system relations at the endemic equilibrium point of the system (3), we arrive at the following conclusion:

$$\dot{V} = \left(\mu_{S_1}S_1^* + (\beta_{1,1}I_1^* + \beta_{1,2}I_2^*)S_1^* - \mu_{S_1}S_1\frac{S_1^*}{S_1} - \left(\mu_{S_1}S_1^* + (\beta_{1,1}I_1^* + \beta_{1,2}I_2^*)\right)\frac{S_1^*}{S_1} + \mu_{S_1}S_1^* + S_1^*(\beta_{1,1}I_1 + \beta_{1,2}I_2)\right) + \left(\mu_{S_2}S_2^* + (\beta_{2,1}I_1^* + \beta_{2,2}I_2^*)S_2^* - \mu_{S_2}S_2\frac{S_2^*}{S_2} - (\mu_{S_2}S_2^* + (\beta_{2,1}I_1^* + \beta_{2,2}I_2^*)S_2^*)\frac{S_2^*}{S_2} + \mu_{S_2}S_2^* + S_2^*(\beta_{2,1}I_1 + \beta_{2,2}I_2)\right) - (\mu_{E_I} + \gamma + \mu_{E_C} + \delta)I_1 - \beta_{1,1}I_1^*S_1\frac{S_1^*}{S_1} - \beta_{1,2}I_1^*S_1\frac{S_1^*}{S_1}\frac{I_1^*}{I_1}\frac{I_2}{I_2} + \beta_{2,1}I_1^*S_2\frac{S_2^*}{S_2} - \beta_{2,2}I_2^*S_2\frac{S_2^*}{S_2}\frac{I_1^*}{I_1}\frac{I_2}{I_2} + \beta_{1,2}I_1^*S_1^* + \beta_{1,2}I_2^*S_2^* + \beta_{2,2}I_2^*S_2^* + \frac{\beta_{1,2}S_1^* + \beta_{2,2}S_2^*}{\mu_I + \gamma_1 + \mu_C + \gamma_2}\left((\delta + \gamma)I_1 - (\mu_I + \gamma_1 + \mu_C + \gamma_2)I_2 - I_2^*(\delta + \gamma)\frac{I_1}{I_2} - (\delta + \gamma)I_1^*\right)$$

$$= \mu_{S_1}S_1^*\left(2 - \frac{S_1^*}{S_1} - \frac{S_1}{S_1}\right) + \mu_{S_2}S_2^*\left(2 - \frac{S_2^*}{S_2} - \frac{S_2}{S_2}\right) + \beta_{1,1}I_1^*S_1^*\left(2 - \frac{S_1^*}{S_1} - \frac{S_1}{S_1}\right) + \beta_{1,2}I_2^*S_1^*\left(2 - \frac{S_1^*}{S_1} - \frac{S_1^*}{S_1}\frac{I_1^*}{I_1}\frac{I_2}{I_2}\right) + \beta_{2,1}I_1^*S_2^*\left(2 - \frac{S_2^*}{S_2} -\right.$$

$$\frac{S_2}{S_2}\Big) + \beta_{2,2}I_2^*S_2^*\left(2 - \frac{S_2^*}{S_2} - \frac{S_2}{S_2}\frac{I_1^*}{I_1}\frac{I_2}{I_2}\right) + \left(\beta_{1,1}S_1^* + \beta_{2,1}S_2^* + \frac{\beta_{1,2}S_1^*+\beta_{2,2}S_2^*}{\mu_I+\gamma_1+\mu_C+\gamma_2}(\delta+\gamma) - \left(\mu_{E_I}+\gamma+\mu_{E_C}+\delta\right)\right)I_1 + \left(\beta_{1,2}S_1^* + \beta_{2,2}S_2^* - \frac{\beta_{1,2}S_1^*+\beta_{2,2}S_2^*}{\mu_I+\gamma_1+\mu_C+\gamma_2}(\mu_I+\gamma_1+\mu_C+\gamma_2)\right)I_2 - \frac{\beta_{1,2}S_1^*+\beta_{2,2}S_2^*}{\mu_I+\gamma_1+\mu_C+\gamma_2}(\delta+\gamma)I_1^*\frac{I_1}{I_1}\frac{I_2^*}{I_2} + + \frac{\beta_{1,2}S_1^*+\beta_{2,2}S_2^*}{\mu_I+\gamma_1+\mu_C+\gamma_2}(\delta+\gamma)I_1^*$$

By posing $\alpha = \frac{\beta_{1,2}S_1^*+\beta_{2,2}S_2^*}{\mu_I+\gamma_1+\mu_C+\gamma_2}$,
it just $\beta_{1,2}S_1^* + \beta_{2,2}S_2^* = \alpha(\mu_I+\gamma_1+\mu_C+\gamma_2)$

$$\Leftrightarrow \beta_{1,2}S_1^* + \beta_{2,2}S_2^* - \alpha(\mu_I+\gamma_1+\mu_C+\gamma_2) = 0 \text{ and } \beta_{1,1}S_1^* + \beta_{2,1}S_2^* + \alpha(\delta+\gamma) - \left(\mu_{E_I}+\gamma+\mu_{E_C}+\delta\right)$$

$$= \frac{\left(\beta_{1,1}S_1^*+\beta_{2,1}S_2^* + \frac{\beta_{1,2}S_1^*+\beta_{2,2}S_2^*}{\mu_I+\gamma_1+\mu_C+\gamma_2}(\delta+\gamma)\right)\left(\mu_{E_I}+\gamma+\mu_{E_C}+\delta\right)}{\left(\mu_{E_I}+\gamma+\mu_{E_C}+\delta\right)} - \left(\mu_{E_I}+\gamma+\mu_{E_C}+\delta\right)$$

$$= \left(\mu_{E_I}+\gamma+\mu_{E_C}+\delta\right)\left(\frac{\left((\beta_{1,1}S_1^*+\beta_{2,1}S_2^*)(\mu_I+\gamma_1+\mu_C+\gamma_2)+(\beta_{1,2}S_1^*+\beta_{2,2}S_2^*)(\delta+\gamma)\right)}{\left(\mu_{E_I}+\gamma+\mu_{E_C}+\delta\right)(\mu_I+\gamma_1+\mu_C+\gamma_2)} - 1\right) = 0$$

In order to establish the relationships set out in (3),

$$0 = \left(\beta_{1,1}I_1^* + \beta_{1,2}I_2^*\right)S_1^* + \left(\beta_{2,1}I_1^* + \beta_{2,2}I_2^*\right)S_2^* - \left(\mu_{E_I}+\gamma+\mu_{E_C}+\delta\right)I_1^*$$

we have: $\left(\beta_{1,1}I_1^* + \beta_{1,2}I_2^*\right)S_1^* + \left(\beta_{2,1}I_1^* + \beta_{2,2}I_2^*\right)S_2^* = \left(\mu_{E_I}+\gamma+\mu_{E_C}+\delta\right)I_1^*$

$$\Leftrightarrow \beta_{1,1}I_1^*S_1^* + \beta_{1,2}\frac{(\delta+\gamma)}{(\mu_I+\gamma_1+\mu_C+\gamma_2)}I_1^*S_1^* + \beta_{2,1}I_1^*S_2^* + \beta_{2,2}\frac{(\delta+\gamma)}{(\mu_I+\gamma_1+\mu_C+\gamma_2)}I_1^*S_2^* = \left(\mu_{E_I}+\gamma+\mu_{E_C}+\delta\right)I_1^*$$

$$\Leftrightarrow \frac{(\mu_I+\gamma_1+\mu_C+\gamma_2)\left(\beta_{1,1}S_1^* + \beta_{2,1}S_2^*\right) + (\delta+\gamma)\left(\beta_{1,2}S_1^* + \beta_{2,2}S_2^*\right)}{(\mu_I+\gamma_1+\mu_C+\gamma_2)\left(\mu_{E_I}+\gamma+\mu_{E_C}+\delta\right)} = 1$$

$$\alpha(\delta+\gamma)I_1^* = \frac{\beta_{1,2}S_1^*+\beta_{2,2}S_2^*}{\mu_I+\gamma_1+\mu_C+\gamma_2}(\delta+\gamma)I_1^*$$

$$= \frac{(\delta+\gamma)}{\mu_I+\gamma_1+\mu_C+\gamma_2}I_1^*\left(\beta_{1,2}S_1^* + \beta_{2,2}S_2^*\right)$$

$$= \beta_{1,2}I_2^*S_1^* + \beta_{2,2}I_2^*S_2^*$$

By employing these relationships in the expression of $\dot{V}$, we arrive at the following equation:

$$\dot{V} = \mu_{S_1}S_1^*\left(2 - \frac{S_1^*}{S_1} - \frac{S_1}{S_1}\right) + \mu_{S_2}S_2^*\left(2 - \frac{S_2^*}{S_2} - \frac{S_2}{S_2}\right) + \beta_{1,1}I_1^*S_1^*\left(2 - \frac{S_1^*}{S_1} - \frac{S_1}{S_1}\right) + \beta_{1,2}I_2^*S_1^*\left(3 - \frac{S_1^*}{S_1} - \frac{S_1^*}{S_1}\frac{I_1^*}{I_1}\frac{I_2}{I_2} - \frac{I_1^*}{I_1}\frac{I_2}{I_2}\right) + \beta_{2,1}I_1^*S_2^*\left(2 - \frac{S_2^*}{S_2} - \frac{S_2}{S_2}\right) + \beta_{2,2}I_2^*S_2^*\left(3 - \frac{S_2^*}{S_2} - \frac{S_2}{S_2}\frac{I_1^*}{I_1}\frac{I_2}{I_2} - \frac{I_1}{I_1}\frac{I_2^*}{I_2}\right) \leq 0$$

V is a strict Lyapunov function, and according to Lyapunov's theorem, the endemic equilibrium point $(S_1^*, S_2^*, I_1^*, I_2^*, R^*)$ is globally asymptotically stable.

3. Numeric Simulation

The digital simulation of the Respiratory Disease Transmission Model around the Figuil Cement Works is designed to investigate the dynamics of respiratory disease transmission influenced by the plant's pollutant emissions. A system of differential equations is employed to model the transitions between different categories of the population, including susceptible, exposed, infectious, chronic and recovered individuals. The objective is to assess the impact of pollution on the health of local residents and to determine the epidemic potential as a function of the baseline reproduction rate R_0. This analysis will facilitate an understanding of the health impact of the cement plant and the development of appropriate control measures.

Table 1. *Estimated parameter values for the model (1) using data for the Regional Delegation of the Environment in the North Region of Cameroon.*

Value (R_0>1)	Value (R_0<1)	Parameters	Value (R_0>1)	Value (R_0<1)	Parameters
0.025	0.02	$\beta_{1,1}$	1	1	Λ
0.045	0.061	$\beta_{1,2}$	0.5	0.5	p_1
0.076	0.05	$\beta_{2,1}$	0.5	0.5	p_2
0.085	0.061	$\beta_{2,2}$	0.01	0.01	μ_{S_1}
0.1	0.05	γ_1	0.01	0.01	μ_{S_2}
0.1	0.05	γ_2	0.025	0.015	μ_{E_I}
0.15	0.1	Δ	0.043	0.085	μ_{E_C}
0.1	0.1	Γ	0.017	0.013	μ_I
1.0066	0.6783	R_0	0.013	0.012	μ_C
			0.1	0.1	μ_R

3.1. Simulate if R_0<1

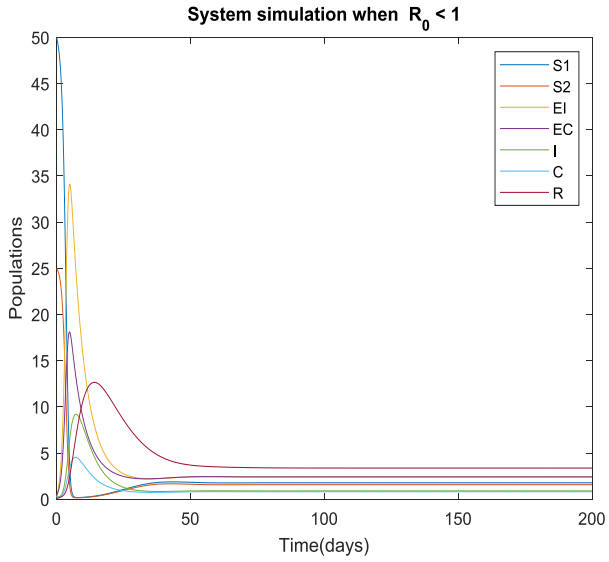

Figure 2. *The system's behaviour in response to R_0=0.6783.*

The system's behaviour can be described as follows: When R_0 is less than one, the model demonstrates a gradual decline in the population of infectious individuals (i.e., $E_I(t)$, I(t), C(t)). Individuals infected or in the latent phase of infection tend to disappear over time.

Stabilisation: The system tends to stabilise with a reduction in the number of cases of infection to very low or zero levels. The susceptible compartments $S_1(t)$ and $S_2(t)$ remain relatively high, as the infection is not spreading significantly in the population.

3.2. Simulate if R_0>1

The system's behaviour can be described as follows: In the event that R_0 is greater than unity, the population of infectious individuals will increase at the outset of the simulation. The growth of $E_I(t)$, I(t), and C(t) indicates the active spread of infection.

The spread of infection is as follows: As a consequence of the elevated value of R_0, a greater proportion of susceptible populations, namely $S_1(t)$ and $S_2(t)$, become infected over time. This results in a reduction in the aforementioned susceptible compartments and an increase in the infectious compartments.

Epidemiological interpretation: When R_0 is greater than one, the infection has the potential to spread significantly, resulting in an epidemic. The epidemic persists, and the number of infectious cases remains high.

Implications for the Control of Epidemics: In the context of public health, it is imperative to control R_0 values below 1 in order to prevent the development of an epidemic. Such measures may include vaccination, social distancing, or other control strategies designed to reduce transmission.

System Stability: The simulation also demonstrates that the initial conditions and model parameters influence the stability of the system. When R_0 < 1, the system reaches a steady state with minimal infection. Conversely, when R_0 > 1, the system can enter into continuous epidemic dynamics if no measures are taken to reduce R_0.

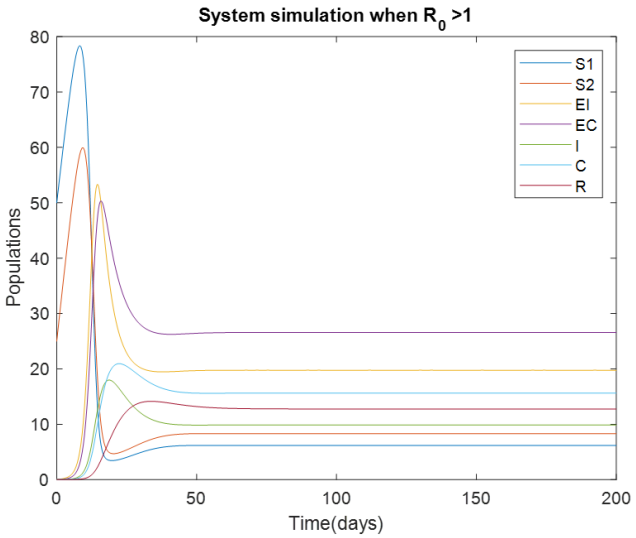

Figure 3. *The system's behaviour in response to R_0=1.0066.*

4. Conclusions

This research demonstrates the considerable impact of atmospheric pollution, particularly the emissions of fine particles and harmful gases from the Figuil Cement, on the prevalence of respiratory diseases among local populations. The

analysis, conducted using a modified SEIR epidemiological model, revealed that elevated pollution levels can exacerbate the transmission of diseases such as asthma and chronic bronchitis, particularly among vulnerable demographic groups, including children and the elderly. The simulation results demonstrate the impact of R_0. When R_0 is less than one, the model predicts a reduction in infection cases and a stabilisation of the system with minimal infection. Conversely, if R_0 is greater than 1, the epidemic can develop continuously, thereby underscoring the necessity for interventions aimed at reducing R_0. The results of the simulations demonstrate that the stability of the system is contingent upon the initial conditions and model parameters. Modifications to transmission rates, recovery rates, and environmental conditions have the potential to impact epidemic dynamics. A balance is reached when R_0 is controlled below 1, thereby facilitating the limitation of the impact of pollution on the spread of diseases. In order to mitigate the harmful effects of pollution on respiratory health, it is recommended that control strategies be implemented, including: This study emphasises the necessity of an integrated strategy for the control of respiratory diseases exacerbated by pollution, which should combine environmental control measures with adapted public health interventions. The simulation of epidemiological models, taking into account the effects of pollution, offers valuable insights for the development of effective and adapted strategies for the reduction of health risks in affected areas.

Abbreviations

NOx	Nitrogen Oxides
NO_2	Nitrogen Dioxide
SO_2	Sulfur Dioxide
PM2.5	Particulate Matter with a Diameter of 2.5 Micrometers
PM10	Particulate Matter with a Diameter of 10 Micrometers
SEIR	Susceptible -Exposed-Infected-Recovered
COPD	Chronic Obstructive Pulmonary Disease
WHO	World Health Organisation
DFE	Disease Free Equilibrium

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Data Availability Statement

The data supporting the findings of this study were collected from the Regional Delegation of the Environment in the North Region of Cameroon.

Conflicts of Interest

The authors declare no conflicts of interest.

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Research Field

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