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ASSESSMENT OF INTERVENTION STRATEGIES TO MITIGATE THE IMPACT OF ENVIRONMENTAL POLLUTION ON ASTHMA PATIENTS USING MATHEMATICAL MODELLING APPROACH

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ABSTRACT

Asthma is a long-term respiratory illness marked by airway inflammation and obstruction, and its severity is often worsened by environmental pollutants such as particulate matter, tobacco smoke. In this study, a deterministic compartmental model was formulated using ordinary differential equations (ODEs) to examine and predict the progression and management of asthma in environments affected by pollution. The model categorizes the population into several health and exposure groups: susceptible (S), exposed (E), infected (I)—with subcategories for mild, moderate, and chronic conditions—recovered (R), and an environmental pollution class (P). The framework also integrates intervention strategies such as health education, pollution control policies, and medical treatment to evaluate their effectiveness in limiting disease spread. The basic reproduction number (Ro) was computed to identify the conditions under which asthma either persists or can be eliminated. The results demonstrated that elevated pollution levels, higher exposure rates, and inadequate interventions significantly increase asthma incidence.

1. INTRODUCTION

Asthma is a prevalent and long-term respiratory illness that affects over 262 million people worldwide and more than 455,000 deaths annually (World Health Organization,2023).[1]. It presents major public health concerns by disrupting daily life and placing a strain on healthcare systems (Global Initiative for Asthma,2023). [2]. Its hallmark is chronic inflammation of the airways, leading to increased airway responsiveness and a tendency for the bronchial tubes to narrow. The causes of asthma are multifactorial, involving both inherited genetic traits and external environmental triggers. While genetic predisposition may make some individuals more vulnerable, factors like airborne allergens, tobacco smoke, air pollutants, respiratory infections, and occupational exposures are well-established in worsening or triggering symptoms (Dharmage et al., 2019; Busse & Lemanske, 2001) [3].

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The manifestation of asthma differs significantly from person to person, with some individuals experiencing occasional, mild symptoms, while others endure persistent and more severe forms of the condition. Typically, asthma presents with recurrent wheezing—a high-pitched whistling sound during breathing—persistent coughing (often more noticeable at night or early in the morning), chest tightness, and episodes of breathlessness. These symptoms are frequently provoked by a range of environmental and physiological triggers, including allergens such as pollen, dust mites, and pet dander; respiratory infections of viral or bacterial origin; physical exertion; exposure to cold air; air pollutants like smoke, ozone, and particulate matter; and certain occupational irritants, including chemicals and industrial fumes [4]. This research work developed a deterministic compartmental model using ordinary differential equations (ODEs). The model divided the human population into various health and exposure categories: susceptible (S), exposed (E), infected (I) with sub-compartments (mild, moderate, chronic), recovered (R), and a dynamic environmental pollution class (P). Additionally, the model incorporated control strategies, such as public health awareness, pollution regulation and medical interventions, to assess their impact on disease transmission and burden [5]. The remainder of this paper is structured as follows: Section 2 presents the model formulation, including all compartments and transmission pathways. Section 3 analyzes the sub-models for the deterministic nature of the dynamics of asthma progression and treatment Determine the Disease-Free Equilibrium (DFE) and Endemic Equilibrium Point (EEP) of the model, we derive the basic reproduction numbers and solving the model equations analytically or numerically. While Section 4 presents numerical simulations and discussion.

2.0 MATERIAL AND METHODS

2.1 Model Variables and Parameters

In Table 2.1 below we present the description of the model variables and parameters.

Table 2.1: Description of Variables and Parameters

Variables	Description
$S(t)$	Number of susceptible individuals at time t
$E(t)$	Number of exposed individual (exposed to pollution) at time t
$I_1(t)$	Number of infected individuals with mild asthma at time t
$I_2(t)$	Number of infected individuals with moderate asthma at time t
I_3(t)	Number of infected individuals with severe asthma at time t
I_4(t))	Number of infected individuals with chronic asthma at time t
$R(t)$	Number of recovered individuals receiving treatment at time t
$P(t)$	Cumulative concentration of pollution in the environment at time t
$N(t)$	Total population at time t
Parameters	**Description**
Λ	Recruitment rate of individuals to the susceptible class.
γ	Transmission rate due to interaction of susceptible with pollutants present in the environment.
λ	The transmission rate at which exposed individuals become infected due to environmental pollutant.
β_1	Progression rate at which individuals with mild asthma progress to moderate asthma.
β_2	Progression rate at which individuals with moderate asthma progress to severe asthma.
β_3	Progression rate at which individuals with severe asthma progress to chronic asthma.
α_1	The average rate of asthma control for individuals with mild asthma
α_2	The average rate of asthma control for individuals with moderate asthma

α_3	The average rate of asthma control for individuals with severe asthma
α_4	The average rate of asthma control for individuals with chronic asthma
σ_i	Asthma induced death rate, where $i = 1, 2, \ldots 5$ represent the concerned compartments.
ϖ	The probability of achieving successful asthma control due to public awareness $(0 < \omega \leq 1)$
τ	The rate at which human activities contribute to the reduction of pollutants in the environment $(0 < \tau \leq 1)$.
δ	The natural attenuation rate of pollutants
μ	Natural death rate
Q	The aggregate emission rate of pollutants from various sources

2.3 Description of Model Formulation

A modified mathematical model was developed to account for the underlying causes of asthma, with particular emphasis on the impact of environmental pollutants as key contributing factors. The total population is divided into eight classes namely; susceptible individuals $S(t)$, exposed individual $E(t)$, infected individual with mild asthma $I_1(t)$, infected individual with moderate asthma $I_2(t)$, infected individual with severe asthma $I_3(t)$, infected individuals with chronic asthma $I_4(t)$, individuals in the recovered class $R(t)$, and an environmental pollution class $P(t)$. sThe recruitment rate into the susceptible population $S(t)$ is given by Λ. It is assumed that the susceptible population become asthmatic when continuously being exposed to pollutant present in the environment in two steps. First individual from the susceptible class move to the exposed class $E(t)$ at a transmission rate γ, and then from the exposed class to the initial infected class $I_1(t)$ at a transmission rate λ due to continuous exposure with environmental pollution. Individual infected with mild asthma $I_1(t)$ can progress into moderate asthma $I_2(t)$ class at a rate β_1. Similarly individual with moderate asthma if not treated or control can progress into the severe asthma class $I_3(t)$ at the rate β_2. Individual with severe asthma can progress into a chronic asthma class $I_4(t)$ at the rate β_3.

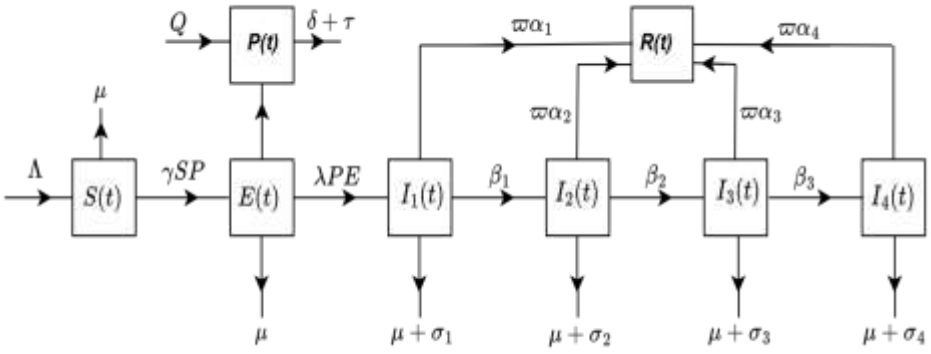

Figure 2.1 Flow diagram of the Mathematical model of Asthma Control

Since asthma disease progresses through four stages, treatment of infected individuals can take place at any of these stages at a rate α_i, the average rate of asthma control, where $i = 1,2,3,4$ is the different stages of asthma progression, moving the infected individual into the recovered class $R(t)$, $P(t)$ is the cummulative concentration of pollution present in the environment at time *t.* We also observed that Q is the rate of emission of pollutant from various sources into the environment. δ is the natural attenuation rate of pollutants which refers to the rate at which environmental pollutants naturally break down, degrade, or are removed from the environment

without human intervention and τ is the depletion rate coefficient of the pollutant concentration in the environment due to human activities.

2.4 Model Equations

Below is the system of nonlinear ordinary differential equations that describe the asthma disease model:

$$S' = \Lambda - \mu S - \gamma SP \quad (2.1)$$

$$E' = \gamma SP - \lambda PE - \mu E \quad (2.2)$$

$$I_1' = \lambda PE - (\mu + \sigma_1)I_1 - \beta_1 I_1 - \varpi\alpha_1 I_1 \quad (2.3)$$

$$I_2' = \beta_1 I_1 - (\mu + \sigma_2)I_2 - \beta_2 I_2 - \varpi\alpha_2 I_2 \quad (2.4)$$

$$I_3' = \beta_2 I_2 - (\mu + \sigma_3)I_3 - \beta_3 I_3 - \varpi\alpha_3 I_3 \quad (2.5)$$

$$I_4' = \beta_3 I_3 - (\mu + \sigma_4)I_4 - \varpi\alpha_4 I_4 \quad (2.6)$$

$$R_4' = \varpi\alpha_4 I_4 + \varpi\alpha_3 I_3 + \varpi\alpha_2 I_2 + \varpi\alpha_1 I_1 - (\mu + \sigma_5)R \quad (2.7)$$

$$P' = Q - (\delta + \tau)P \quad (2.8)$$

$$N(t) = S(t) + E(t) + I_1(t) + I_2(t) + I_3(t) + I_4(t) + R(t) + P(t) \quad (2.9)$$

where $N(t)$ is the total population at time *t*

2.5 Positivity and Boundedness of Solution of the Asthma Model

We would show that the solution to the model equations is epidemiological and mathematically well posed and is positive in the invariant region of attraction Ω. We obtained the invariant set Ω, where the model solution is bounded.

Theorem 2.1

If $S(0), E(0), I_1(0), I_2(0), I_3(0), I_4(0), R(0), P(0)$ are non-negative, the solution $S(t), E(t), I_1(t), I_2(t), I_3(t), I_4(t), R(t), P(t)$ of the Asthma disease model are non-negative for all $t \geq 0$

Proof:

From equation (2.9), we have that

$$N'(t) = S'(t) + E'(t) + I_1'(t) + I_2'(t) + I_3'(t) + I_4'(t) + R'(t) + P'(t) \quad (2.10)$$

Substituting equations (2.1) to (2.8) into equation (2.10) we have,

$$N'(t) = \Lambda - (S + E + I_1 + I_2 + I_3 + I_4 + R)\mu - (\sigma_1 I_1 + \sigma_2 I_2 + \sigma_3 I_3 + \sigma_4 I_4 + \sigma_5 R) + Q - (\delta + \tau)P \quad (2.11)$$

Setting the disease induce death rate to be zero $(\sigma_1 = \sigma_2 = \sigma_3 = \sigma_4 = \sigma_5 = 0)$ and let $N(t) = S + E + I_1 + I_2 + I_3 + I_4 + R$, we have

$$N'(t) = \Lambda - \mu N + Q - (\delta + \tau)P \quad (2.12)$$

$$N'(t) \leq \Lambda - \mu N \quad (2.13)$$

Solving equation (2.13) by method of separation of variable, we have

$$\frac{dN}{dt} \leq \Lambda - \mu N \implies \frac{dN}{\Lambda - \mu N} \leq dt \quad (2.14)$$

Integrating both side of equation (3.14), we have

$-\frac{1}{\mu} In(\Lambda - \mu N) \leq t + K \implies In(\Lambda - \mu N) \geq -\mu(t + K)$

$\implies |\Lambda - \mu N| \geq Ae^{-\mu(t+K)} \implies |\Lambda - \mu N| \geq Ae^{-\mu t}$ (2.15)

where *A* is the constant of integration.

Applying the initial condition $N(0) = N_0$ to equation (2.15), we have that

$\Lambda - \mu N_0 = A$ (2.16)

Substituting equation (2.16) into equation (2.15) we have

$\Lambda - \mu N \geq (\Lambda - \mu N_0)e^{-\mu t}$ (2.17)

Rearranging equation (2.17), we have

$N \leq \frac{\Lambda}{\mu} - \left(\frac{\Lambda - \mu N_0}{\mu}\right) e^{-\mu t}$ (2.18)

As $t \longrightarrow \infty$ in equation (2.18), the population size at time $N = \frac{\Lambda}{\mu} \left(0 \leq N \leq \frac{\Lambda}{\mu}\right)$.

This implies that the feasible solution set of the nonlinear system of equation of the model enter and remain in the region Ω

$\Omega = \left\{ (S(t), E(t), I_1(t), I_2(t), I_3(t), I_4(t), R(t)) \in \mathcal{R}^7, N \leq \frac{\Lambda}{\mu} \right\}$ (2.19)

Therefore, all feasible solutions of the system of differential equations (2.1) to (2.8) enter the region. The region Ω is positively invariant with respect to the model equation.

2.6 Disease - Free Equilibrium of Asthma Disease Model (E_o)

At the Disease-Free Equilibrium, we make the assumption that the disease compartment variables become zero, hence $E(t) = I_1(t) = I_2(t) = I_3(t) = I_4(t) = R(t) = 0$ and that. Hence $E_0 = (S(t), 0,0,0,0,0,0, P(t))$

In the system of nonlinear differential equation given in equation (2.1) - (2.8), we set the derivative to be equal to zero and the equation becomes

$0 = \Lambda - \mu S - \gamma SP \implies S = \frac{\Lambda}{\mu + \gamma P}$ (2.20)

$0 = Q - (\delta + \tau)P \implies P(t) = \frac{Q}{\delta + \tau}$ (2.21)

Therefore, the disease-free equilibrium is

$E_o = \left(\frac{\Lambda}{\mu + \gamma P}, 0, 0, 0, 0, 0, 0, \frac{Q}{\delta + \tau}\right)$ (2.22)

2.7 Existence of Endemic Equilibrium of Asthma Disease Model (E_0^*)

Here we assume that the Asthma Disease due to environmental pollution in the population will persist with time t and hence $S(t) \neq E(t) \neq I_1(t) \neq I_2(t) \neq I_3(t) \neq I_4(t) \neq R(t) \neq P(t) \neq 0$. Also, we know that $\lambda = 1, \gamma = 1$ and $\delta = 0$

From equation (2.8), we have

$P' = Q - (\delta + \tau)P = 0$

$\Rightarrow' Q - \tau P = 0$

$\Rightarrow P(t) = \frac{Q}{\tau}$ (2.23)

From equation (2.1), we have that

$0 = \Lambda - \mu S - \gamma SP \Rightarrow \Lambda - (\mu + P)S = 0 \implies \Lambda = (\mu + P)S$

$\implies S = \frac{\Lambda}{\mu + P} = \frac{\Lambda}{\mu + \frac{Q}{\tau}} = \frac{\Lambda \tau}{\mu \tau + Q}$ (Substitute equation (2.23) and simplify)

$\implies S(t) = \frac{\Lambda \tau}{\mu \tau + Q}$ (2.24)

From equation (2.2), we have that

$0 = \gamma SP - \lambda PE - \mu E \implies 0 = SP - PE - \mu E \implies 0 = SP - (P + \mu)E$

Substituting $S(t)$ and $P(t)$, we have

$\Rightarrow 0 = \left(\frac{\Lambda\tau}{\mu\tau+Q}\right)\left(\frac{Q}{\tau}\right) - \left(\frac{Q}{\tau}+\mu\right)E \quad \Rightarrow 0 = \left(\frac{\Lambda\tau}{\mu\tau+Q}\right)\left(\frac{Q}{\tau}\right) - \left(\frac{Q+\mu\tau}{\tau}\right)E$

$\Rightarrow 0 = \left(\frac{\Lambda\tau}{\mu\tau+Q}\right)\left(\frac{Q}{\tau}\right) = \left(\frac{Q+\mu\tau}{\tau}\right)E \Rightarrow E(t) = \left(\frac{\Lambda\tau}{\mu\tau+Q}\right)\left(\frac{Q}{\tau}\right)\cdot\left(\frac{\tau}{Q+\mu\tau}\right)$

$\Rightarrow E(t) = \left(\frac{\Lambda\tau}{\mu\tau+Q}\right)\left(\frac{Q}{\mu\tau+Q}\right)$

$\Rightarrow E(t) = \frac{\Lambda\tau Q}{(\mu\tau+Q)^2}$ (2.25)

From equation (2.3), we have that

$\Rightarrow 0 = \lambda PE - (\mu+\sigma_1)I_1 - (\beta_1+\varpi\alpha_1)I_1 \Rightarrow 0 = PE - (\mu+\sigma_1)I_1 - (\beta_1+\varpi\alpha_1)I_1$

$\Rightarrow 0 = PE - (\mu+\sigma_1+\beta_1+\varpi\alpha_1)I_1 \Rightarrow PE = (\mu+\sigma_1+\beta_1+\varpi\alpha_1)I_1$

$\Rightarrow I_1 = \frac{PE}{\mu+\sigma_1+\beta_1+\varpi\alpha_1}$

Substituting $S(t)$ and $E(t)$, we have

$\Rightarrow I_1 = \left(\frac{Q}{\tau}\right)\left(\frac{\Lambda\tau Q}{(\mu\tau+Q)^2}\right)\left(\frac{1}{\mu+\sigma_1+\beta_1+\varpi\alpha_1}\right)$

$I_1(t) = \frac{\Lambda Q^2}{(\mu\tau+Q)^2(\mu+\sigma_1+\beta_1+\varpi\alpha_1)}$ (2.26)

From equation (2.4), we have that

$\Rightarrow 0 = \beta_1 I_1 - (\mu+\sigma_2)I_2 - (\beta_2+\varpi\alpha_2)I_2 \Rightarrow 0 = \beta_1 I_1 - (\mu+\sigma_2+\beta_2+\varpi\alpha_2)I_2$

$\Rightarrow I_2 = \frac{\beta_1 I_1}{\mu+\sigma_2+\beta_2+\varpi\alpha_2}$

Substituting $I_1(t)$, we have

$I_2(t) = \frac{\beta_1\Lambda Q^2}{(\mu+\sigma_2+\beta_2+\varpi\alpha_2)(\mu\tau+Q)^2(\mu+\sigma_1+\beta_1+\varpi\alpha_1)}$ (2.27)

From equation (2.5), we have that

$\Rightarrow 0 = \beta_2 I_2 - (\mu+\sigma_3)I_3 - (\beta_3+\varpi\alpha_3)I_3 \Rightarrow \beta_2 I_2 = (\mu+\sigma_3+\beta_3+\varpi\alpha_3)I_3$

$\Rightarrow I_3 = \frac{\beta_2 I_2}{\mu+\sigma_3+\beta_3+\varpi\alpha_3}$

Substituting $I_2(t)$, we hav2

$I_3(t) = \frac{\beta_2\beta_1\Lambda Q^2}{(\mu+\sigma_3+\beta_3+\varpi\alpha_3)(\mu+\sigma_2+\beta_2+\varpi\alpha_2)(\mu\tau+Q)^2(\mu+\sigma_1+\beta_1+\varpi\alpha_1)}$ (2.28)

From equation (2.6), we have that

$\Rightarrow 0 = \beta_3 I_3 - (\mu+\sigma_4)I_4 - \varpi\alpha_4 I_4 \Rightarrow \beta_3 I_3 = (\mu+\sigma_4+\varpi\alpha_4)I_4$

$\Rightarrow I_4 = \frac{\beta_3 I_3}{\mu+\sigma_4+\varpi\alpha_4}$

Substituting $I_3(t)$, we have

$I_4(t) = \frac{\beta_3\beta_2\beta_1\Lambda Q^2}{(\mu+\sigma_4+\varpi\alpha_4)(\mu+\sigma_3+\beta_3+\varpi\alpha_3)(\mu+\sigma_2+\beta_2+\varpi\alpha_2)(\mu\tau+Q)^2(\mu+\sigma_1+\beta_1+\varpi\alpha_1)}$ (2.29)

From equation (2.7), we have that

$\Rightarrow 0 = \varpi\alpha_4 I_4 + \varpi\alpha_3 I_3 + \varpi\alpha_2 I_2 + \varpi\alpha_1 I_1 - (\mu+\sigma_5)R$

$\Rightarrow \varpi\alpha_4 I_4 + \varpi\alpha_3 I_3 + \varpi\alpha_2 I_2 + \varpi\alpha_1 I_1 = (\mu+\sigma_5)R$

$\Rightarrow R(t) = \frac{\varpi\alpha_4 I_4+\varpi\alpha_3 I_3+\varpi\alpha_2 I_2+\varpi\alpha_1 I_1}{\mu+\sigma_5}$

Substituting $I_1(t), I_2(t), I_3(t)$ and $I_4(t)$, we have

$R(t) = \frac{\varpi\Lambda Q^2}{W}\left(\frac{\alpha_1}{N^2M_1} + \frac{\alpha_2\beta_1}{N^2M_1M_2} + \frac{\alpha_3\beta_1\beta_2}{N^2M_1M_2M_3} + \frac{\alpha_4\beta_1\beta_2\beta_3}{N^2M_1M_2M_3M_4}\right)$

were

$$\left.\begin{array}{ll} N = \mu\tau + Q, & M_2 = \mu+\sigma_2+\beta_2+\varpi\alpha_2 \\ W = \mu+\sigma_5, & M_3 = \mu+\sigma_3+\beta_3+\varpi\alpha_3 \\ M_1 = \mu+\sigma_1+\beta_1+\varpi\alpha_1, & M_4 = \mu+\sigma_4+\varpi\alpha_4 \end{array}\right\} \quad (2.30)$$

Therefore, the endemic equilibrium (E_0^*) is given as $E_0^* = (S^*, E^*, I_1^*, I_2^*, I_3^*, I_4^*, R^*, P^*)$

$$E_0^* = \left(\frac{\Lambda\tau}{N}, \frac{\Lambda\tau Q}{N^2}, \frac{\Lambda Q^2}{N^2 M_1}, \frac{\beta_1 \Lambda Q^2}{N^2 M_1 M_2}, \frac{\beta_1\beta_2 \Lambda Q^2}{N^2 M_1 M_2 M_3}, \frac{\beta_1\beta_2\beta_3 \Lambda Q^2}{N^2 M_1 M_2 M_3 M_4}, \frac{\varpi \Lambda Q^2}{W}\left(\frac{\alpha_1}{N^2 M_1} + \frac{\alpha_2\beta_1}{N^2 M_1 M_2} + \frac{\alpha_3\beta_1\beta_2}{N^2 M_1 M_2 M_3} + \frac{\alpha_4\beta_1\beta_2\beta_3}{N^2 M_1 M_2 M_3 M_4}\right), \frac{Q}{\tau}\right)$$

$$S^*(t) = \frac{\Lambda\tau}{\mu\tau+Q} \tag{2.31}$$

$$E^*(t) = \frac{\Lambda\tau Q}{(\mu\tau+Q)^2}$$

$$I_1^*(t) = \frac{\Lambda Q^2}{(\mu\tau+Q)^2(\mu+\sigma_1+\beta_1+\varpi\alpha_1)}$$

$$I_2^*(t) = \frac{\beta_1 \Lambda Q^2}{(\mu+\sigma_2+\beta_2+\varpi\alpha_2)(\mu\tau+Q)^2(\mu+\sigma_1+\beta_1+\varpi\alpha_1)}$$

$$I_3^*(t) = \frac{\beta_2\beta_1 \Lambda Q^2}{(\mu+\sigma_3+\beta_3+\varpi\alpha_3)(\mu+\sigma_2+\beta_2+\varpi\alpha_2)(\mu\tau+Q)^2(\mu+\sigma_1+\beta_1+\varpi\alpha_1)}$$

$$I_4^*(t) = \frac{\beta_3\beta_2\beta_1 \Lambda Q^2}{(\mu+\sigma_4+\varpi\alpha_4)(\mu+\sigma_3+\beta_3+\varpi\alpha_3)(\mu+\sigma_2+\beta_2+\varpi\alpha_2)(\mu\tau+Q)^2(\mu+\sigma_1+\beta_1+\varpi\alpha_1)}$$

$$R^*(t) = \frac{\varpi \Lambda Q^2}{W}\left(\frac{\alpha_1}{N^2 M_1} + \frac{\alpha_2\beta_1}{N^2 M_1 M_2} + \frac{\alpha_3\beta_1\beta_2}{N^2 M_1 M_2 M_3} + \frac{\alpha_4\beta_1\beta_2\beta_3}{N^2 M_1 M_2 M_3 M_4}\right)$$

$$P^*(t) = \frac{Q}{\tau}$$

3.0 COMPUTATION OF THE BASIC REPRODUCTION NUMBER (R_0) OF THE MODEL

The number of secondary infections that one infectious person might cause during the infectious period, assuming that everyone else is susceptible, is known as the Basic Reproductive Number (R_0) The production of secondary cases is insufficient to sustain the infection in the human community below a certain threshold, known as R_0. If R_0 is less than 1, the disease will die out and fewer people will be infected from generation to generation; if R_0 is greater than 1, the disease will continue to exist and more people will be affected.

The Next Generation Matrix (NGM) method, developed by van den Driessche and Watmough (2022), is a standard approach used to compute the basic reproductive number (R_0) in compartmental epidemic models. It works by dividing the system into two main components: *F(x)*, representing the rate of new infections, and *V(x)*, representing the rate of transitions between compartments. Matrices F and V are then constructed using partial derivatives of these components. However, this method is unsuitable for the current asthma–pollution model because the model's infection process depends on an environmental variable (*P*)—pollution—which lies outside the infected subsystem. In this model, new infections are driven by contact between susceptible individuals and environmental pollutants, rather than by contact between infectious individuals, violating a fundamental assumption of the NGM approach that requires infection terms to depend solely on infected compartments.

Furthermore, the environmental variable *P* maintains a nonzero value even at the disease-free equilibrium (DFE), since it is sustained by continuous emissions from human activities. This persistence means that the infection term γSP does not vanish at DFE, making linearization in terms of infected states invalid. Because the NGM framework assumes internal disease transmission—where infection arises endogenously from infected individuals—the exogenous and constant nature of pollution disrupts the feedback structure required for accurate R_0 estimation. Consequently, the NGM method cannot be applied to this model. Instead, a mechanistic derivation of R_0, based on transition probabilities and average duration within disease states, provides a more accurate and epidemiologically meaningful approach for capturing asthma dynamics driven by environmental pollution

3.2 Mechanistic/Pathway-Based Approach for the Computation of R_0

The Mechanistic/Partway-Based Approach is based on the principle of tracking a single infected individual through all disease stages accounting for the probabilities of progression, duration in each stage and the rate of transmission in each stage. This approach is best suited for nonlinear models with exogenous drivers such as environmental pollution, external sources, multiple stages or progression chain. Example of this approach is seen in Martcheva (2015) and van den Driesshe (2007) respectively. [2] The model consider in this work includes external environmental pollution as the infection driver, multiple sequential disease stages and no internal transmission between human, hence the next generation matrix method is not applicable. The Mechanistic/Pathway-Based approach is best appropriate as it track the progression from exposure to chronic infection and sum all contributions to get the R_0.

Using the Mechanistic/Partway-Based Approach, we follow the steps below

Step 1: Define your Infected Subsystem

From our model, the infected compartments are $x = [E \quad I_1 \quad I_2 \quad I_3 \quad I_4]^T$

Step 2: Extract New Infections $\mathcal{F}(x)$

From our model equation, other compartments do not receive new infection. The only new infection term is found in equation (3.1) given below

$E' = \gamma SP - \lambda PE - \mu E$

The only new infection term is $\mathcal{F}_1 = \gamma SP$

Step 3: Define the Progression and Removal Terms $\mathcal{V}(x)$

From the model equation (3.1) - (3.5), selecting only infected compartment, we have

$$E' = \gamma SP - (\lambda P + \mu)E \quad (3.1)$$
$$I_1' = \lambda PE - (\mu + \sigma_1)I_1 - \beta_1 I_1 - \varpi\alpha_1 I_1 \quad (3.2)$$
$$I_2' = \beta_1 I_1 - (\mu + \sigma_2)I_2 - \beta_2 I_2 - \varpi\alpha_2 I_2 \quad (3.3)$$
$$I_3' = \beta_2 I_2 - (\mu + \sigma_3)I_3 - \beta_3 I_3 - \varpi\alpha_3 I_3 \quad (3.4)$$
$$I_4' = \beta_3 I_3 - (\mu + \sigma_4)I_4 - \varpi\alpha_4 I_4 \quad (3.5)$$

The progression and removal terms define by

$$\mathcal{V}(x) = \begin{pmatrix} -(\lambda P + \mu)E \\ \lambda PE - (\mu + \sigma_1 + \beta_1 + \varpi\alpha_1)I_1 \\ \beta_1 I_1 - (\mu + \sigma_2 + \beta_2 + \varpi\alpha_2)I_2 \\ \beta_2 I_2 - (\mu + \sigma_3 + \beta_3 + \varpi\alpha_3)I_3 \\ \beta_3 I_3 - (\mu + \sigma_4 + \varpi\alpha_4)I_4 \end{pmatrix} \quad (3.6)$$

We now compute the expected number of secondary infections by tracking how one exposed individual enters E through γSP, how they progress $E \longrightarrow I_1 \longrightarrow I_2 \longrightarrow I_3 \longrightarrow I_4$ and how long they stay in each compartment.

Step 4: We now Compute R_0 as a Product of Probabilities and Durations.

We now compute the average number of new infections caused by one exposed individual from the susceptible contact through all the disease compartments. The following formula will be used:

$$\text{Duration in the compatment} = \text{Progression and Removal term}^{-1} \quad (3.7)$$
$$\text{Probability to progress} = \frac{\text{Rate of Entering a Compartment}}{\text{Duration in the Compartment}} \quad (3.8)$$

(a). From Susceptible Contact (New Exposure)

At Disease-Free Equilibrium (DFE), we have that $S^* = \frac{\Lambda}{\mu}$ and $P^* = \frac{Q}{\delta+\tau}$

force of infection is given by $\gamma S^* P^* = \gamma \cdot \frac{\Lambda}{\mu} \cdot \frac{Q}{\delta+\tau}$ (3.9)

Equation (3.7) shows how someone enters the exposed compartment E, which is the start of transmission. Now from one exposed individual, we follow their trajectory through the other compartments.

(b). Expected Transition Path from E

(I). From E to I_1

Rate of transition from E to I_1 is λP^*

Duration in E: $\frac{1}{\lambda P^*+\mu}$

Probability to Progress from E to I_1 is $\frac{\lambda P^*}{\lambda P^*+\mu}$

(ii). From I_1 to I_2

Rate of transition from I_1 to I_2 is β_1

Probability to reach I_1 is $\frac{\lambda P^*}{\lambda P^*+\mu}$

Duration in I_1: $\frac{1}{\mu+\sigma_1+\beta_1+\varpi\alpha_1}$

Probability to Progress from I_1 t o I_2 is $\frac{\beta_1}{\mu+\sigma_1+\beta_1+\varpi\alpha_1}$

(iii). From I_2 to I_3

Rate of transition from I_2 to I_3 is β_2

Probability to reach I_2 is $\frac{\beta_1}{\mu+\sigma_1+\beta_1+\varpi\alpha_1}$

Duration in I_2: $\frac{1}{\mu+\sigma_2+\beta_2+\varpi\alpha_2}$

Probability to Progress from I_2 to I_3 is $\frac{\beta_2}{\mu+\sigma_2+\beta_2+\varpi\alpha_2}$

(iv). From I_3 to I_4

Rate of transition from I_3 to I_4 is β_3

Probability to reach I_3 is $\frac{\beta_2}{\mu+\sigma_2+\beta_2+\varpi\alpha_2}$

Duration in I_3: $\frac{1}{\mu+\sigma_3+\beta_3+\varpi\alpha_3}$

Probability to Progress from I_3 to I_4 is $\frac{\beta_3}{\mu+\sigma_3+\beta_3+\varpi\alpha_3}$

Step 5 Multiply all the Probabilities with the Force of Infection

The formula for the Mechanistic/Partway-Based approach for computing R_0 is given below

$$R_0 = \begin{matrix}\text{Force of}\\ \text{Infection at DFE}\end{matrix} \times \begin{matrix}\text{Probability of Progress}\\ \text{from } E \text{ to } I_1\end{matrix} \times \begin{matrix}\text{Probability of Progress}\\ \text{from } I_1 \text{ to } I_2\end{matrix} \times \begin{matrix}\text{Probability of Progress}\\ \text{from } I_2 \text{ to } I_3\end{matrix} \times \begin{matrix}\text{Probability of Progress}\\ \text{from } I_3 \text{ to } I_4\end{matrix} \quad (3.10)$$

According to our model formulation, we have the Reproduction number (R_0) as

$$R_0 = \gamma \cdot S^* \cdot P^* \times \left(\frac{\lambda P^*}{\lambda P^*+\mu}\right) \times \left(\frac{\beta_1}{\mu+\sigma_1+\beta_1+\varpi\alpha_1}\right) \times \left(\frac{\beta_2}{\mu+\sigma_2+\beta_2+\varpi\alpha_2}\right) \times \left(\frac{\beta_3}{\mu+\sigma_3+\beta_3+\varpi\alpha_3}\right) \quad (3.11)$$

where

$\gamma \cdot S^* \cdot P^* = \begin{matrix}\text{Force of}\\ \text{Infection at DFE}\end{matrix}$

$\frac{\lambda P^*}{\lambda P^* + \mu} = \text{Probability of Progress from } E \text{ to } I_1$

$\frac{\beta_1}{\mu+\sigma_1+\beta_1+\varpi\alpha_1}$ = Probability of Progress from I_1 to I_2

$\frac{\beta_2}{\mu+\sigma_2+\beta_2+\varpi\alpha_2}$ = Probability of Progress from I_2 to I_3

$\frac{\beta_3}{\mu+\sigma_3+\beta_3+\varpi\alpha_3}$ = Probability of Progress from I_3 to I_4

Substituting the value at DFE $E_o = \left(\frac{\Lambda}{\mu+\gamma P}, 0, 0, 0, 0, 0, 0, \frac{Q}{\delta+\tau}\right)$ in equation (3.11), we have

$$R_0 = \frac{\gamma\lambda\Lambda Q^2}{(\gamma Q+\mu(\delta+\tau))(\lambda Q+\mu(\delta+\tau))} \times \left(\frac{\beta_1}{M_1}\right) \times \left(\frac{\beta_2}{M_2}\right) \times \left(\frac{\beta_3}{M_3}\right) \quad (3.12)$$

$$R_0 = \frac{\gamma\lambda\Lambda Q^2\beta_1\beta_2\beta_3}{M_1M_2M_3M_6M_7} \quad (3.13)$$

where $M_1 = \mu+\sigma_1+\beta_1+\varpi\alpha_1$, $M_2 = \mu+\sigma_2+\beta_2+\varpi\alpha_2$, $M_3 = \mu+\sigma_3+\beta_3+\varpi\alpha_3$

$M_6 = \gamma Q+\mu(\delta+\tau)$, $M_7 = \lambda Q+\mu(\delta+\tau)$

3.2 Local Stability of Disease -Free Equilibrium (E_0) of the Asthma Disease Model

Theorem 3.1

The disease- free equilibrium (E_0) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. Then the theorem implies that the disease will die out in the community at time t.

Proof:

To prove the local stability of the disease-free equilibrium (E_0), we use the variational matrix obtained at the DFE in section 2.6

Let the variational matrix as in section 2.6.4 given below

$$J_{E_0} = \begin{bmatrix} -\frac{M_6}{M_5} & 0 & 0 & 0 & 0 & 0 & 0 & -\frac{\gamma\Lambda M_5}{M_6} \\ \frac{\gamma Q}{M_5} & -\frac{M_7}{M_5} & 0 & 0 & 0 & 0 & 0 & \frac{\gamma\Lambda M_5}{M_6} \\ 0 & \frac{\lambda Q}{M_5} & -M_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \beta_1 & -M_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \beta_2 & -M_3 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \beta_3 & -M_4 & 0 & 0 \\ 0 & 0 & \varpi\alpha_1 & \varpi\alpha_2 & \varpi\alpha_3 & \varpi\alpha_4 & -W & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -M_5 \end{bmatrix} \quad (3.14)$$

where $M_1 = \mu+\sigma_1+\beta_1+\varpi\alpha_1$, $M_2 = \mu+\sigma_2+\beta_2+\varpi\alpha_2$, $M_3 = \mu+\sigma_3+\beta_3+\varpi\alpha_3$

$M_4 = \mu+\sigma_4+\varpi\alpha_4$, $M_5 = \delta+\tau$, $M_6 = \gamma Q+\mu(\delta+\tau)$, $M_7 = \lambda Q+\mu(\delta+\tau)$ and

$W = \mu+\sigma_5$

The characteristics equation corresponding to the variational matrix (J_{E_0}) is given by

$$\lambda^8 + k_1\lambda^7 + k_2\lambda^6 + k_3\lambda^5 + k_4\lambda^4 + k_5\lambda^3 + k_6\lambda^2 + k_7\lambda + k_8 = 0 \quad (3.15)$$

where $k_1 = \frac{1}{M_5}[WM_5 + M_1M_5 + M_2M_5 + M_3M_5 + M_4M_5 + M_5^2 + M_6 + M_7]$

$$k_2 = \frac{1}{M_5^2}\big[WM_5^2(M_1+M_2+M_3+M_4) + WM_5^3 + M_1M_5^2(M_2+M_3+M_4) + M_1M_5^3 + M_2M_5^2(M_3+M_4) + M_2M_5^3 + M_3\big(M_4M_5^2+M_5^3\big) + M_4M_5^3 + (M_6+M_7)\big(M_2M_5+M_3M_5+M_4M_5+M_5^2+1\big)\big]$$

$$k_3 = \frac{1}{M_5^2}\big[WM_1M_5^2(M_2+M_3) + \big(M_4M_5^2+M_5^3\big)(WM_1+WM_3+M_1M_3+M_2M_3) + \big(M_3M_5^2+M_4M_5^2+M_5^3\big)(WM_2+M_1M_2) + M_4M_5^3(W+M_1+M_2+M_3) + (M_6+M_7)\big(WM_1M_5+WM_2M_5+WM_3M_5+WM_4M_5+WM_5^2+M_1M_2M_5 + M_1M_3M_5+M_1M_4M_5+M_1M_5^2+M_2M_3M_5+M_2M_4M_5+M_2M_5^2+M_3M_4M_5+M_3M_5^2 + M_4M_5^2\big) + (M_6M_7)(W+M_1+M_2+M_3+M_4+M_5)\big]$$

$$k_4 = \frac{1}{M_5^2}[(WM_1M_2M_5^2+WM_1M_5^3)(M_3+M_4) + (M_2+M_3)(WM_4M_5^3+M_1M_4M_5^3) + WM_2M_5^3(M_1+M_3) + WM_3M_4M_5^2(M_1+M_2+M_1M_2) + M_2M_3M_5^3(M_1+M_4) + (M_6+M_7)(WM_1M_2M_5+WM_1M_3M_5+WM_1M_4M_5+WM_1M_5^2+WM_2M_3M_5+WM_2M_4M_5 + WM_2M_5^2+WM_3M_4M_5+WM_3M_5^2+WM_4M_5^2+M_1M_2M_3M_5+M_1M_2M_4M_5+M_1M_2M_5^2 + M_1M_3M_4M_5+M_1M_3M_5^2+M_1M_4M_5^2+M_2M_3M_4M_5+M_2M_3M_5^2+M_2M_4M_5^2+M_3M_4M_5^2) + (M_6M_7)(WM_1+WM_2+WM_3+WM_4+WM_5+M_1M_2+M_1M_3+M_1M_4+M_1M_5+M_2M_3 + M_2M_4+M_2M_5+M_3M_4+M_3M_5+M_4M_5)]$$

$$k_5 = \frac{1}{M_5^2}\big[WM_1M_2M_3\big(M_4M_5^2+M_5^3\big) + WM_1M_4M_5^3(M_2+M_3) + M_2M_3M_4M_5^3(W+M_1) + (M_6+M_7)\big(WM_1M_2M_3M_5+WM_1M_2M_4M_5+WM_1M_2M_5^2+WM_1M_3M_4M_5 + WM_1M_3M_5^2+WM_1M_4M_5^2+WM_2M_3M_4M_5+WM_2M_3M_5^2+WM_2M_4M_5^2 + WM_3M_4M_5^2+M_1M_2M_3M_4M_5+M_1M_2M_3M_5^2+M_1M_2M_4M_5^2+M_1M_3M_4M_5^2 + M_2M_3M_4M_5^2\big) + (M_6M_7)(WM_1M_2+WM_1M_3+WM_1M_4+WM_1M_5+WM_2M_3+WM_2M_4 + WM_2M_5+WM_3M_4+WM_3M_5+WM_4M_5+M_1M_2M_3+M_1M_2M_4+M_1M_2M_3 + M_1M_3M_4+M_1M_3M_5+M_1M_4M_5+M_2M_3M_4+M_2M_3M_5+M_2M_4M_5+M_3M_4M_5)\big]$$

$$k_6 = \frac{1}{M_5^2}\big[WM_1M_2M_3M_4M_5(M_6+M_7) + M_2M_3M_4M_6M_7(W+M_1) + +WM_1M_2M_5\big(M_4M_5^3+M_6M_5^2\big) + WM_1M_2M_5^2(M_3M_7+M_4M_6) + WM_1M_4M_5^2(M_2M_7+M_3M_6) + WM_3M_4M_5^2(M_1M_7+M_2M_6) + WM_2M_3M_4M_5^2(M_7+M_1M_6) + M_1M_2M_7\big(M_3M_4M_5^2+WM_5M_6\big) + WM_5M_6M_7(M_3+M_4)(M_1+M_2) + M_3M_5M_6M_7(WM_4+M_1M_2) + M_4M_6M_7(M_2+M_3)(M_1M_5+WM_1) + M_2M_3M_6M_7(M_4M_5+WM_1)\big]$$

$$k_7 = \frac{1}{M_5^2}\big[WM_1M_2M_3M_4M_5^2(M_6+M_7) + M_6M_7[WM_1M_2M_5(M_3+M_4) + WM_3M_4M_5(M_1+M_2) + M_1M_2M_3M_4(W+M_5)]\big]$$

$$k_8 = \frac{1}{M_5}(WM_1M_2M_3M_4M_6M_7)$$

By using the Routh-Hurwitz criteria, the equilibrium point E_o is locally asymptotically stable if the all the coefficients $k_i > 0$, all the first column of the Routh table are positive and there is no sign changes in the first column.

Theorem 3.2

The disease-free equilibrium (E_0) is locally asymptotically stable if (a). the determinant of the Jacobian matrix (also known as variational matrix) is positive and the trace is negative. (b). on solving the characteristics equation, the roots (eigen values) are all negative.

Proof:

Solving the equation $\left|J_{E_0} - \eta I\right| = 0$, we have

$$\begin{vmatrix} -\frac{M_6}{M_5}-\eta & 0 & 0 & 0 & 0 & 0 & 0 & -\frac{\gamma\Lambda M_5}{M_6} \\ \frac{\gamma Q}{M_5} & -\frac{M_7}{M_5}-\eta & 0 & 0 & 0 & 0 & 0 & \frac{\gamma\Lambda M_5}{M_6} \\ 0 & \frac{\lambda Q}{M_5} & -M_1-\eta & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \beta_1 & -M_2-\eta & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \beta_2 & -M_3-\eta & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \beta_3 & -M_4-\eta & 0 & 0 \\ 0 & 0 & \varpi\alpha_1 & \varpi\alpha_2 & \varpi\alpha_3 & \varpi\alpha_4 & -W-\eta & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -M_5-\eta \end{vmatrix} = 0 \quad (3.16)$$

The Determinant of the Jacobian matrix above is $\left|J_{E_0}\right| = \frac{1}{M_5}(WM_1M_2M_3M_4M_6M_7)$ and

$$\text{Trace} = -\left(M_1 + M_2 + M_3 + M_4 + M_5 + \frac{M_6}{M_5} + \frac{M_7}{M_5} + W\right)$$

Also solving the characteristics equation given in equation (3.15), we have the eigen values as

$\eta_1 = -M_5,\ \ \eta_2 = -\frac{M_7}{M_5},\qquad \eta_3 = -\frac{M_6}{M_5},\qquad \eta_4 = -M_4,\qquad \eta_5 = -M_3,\qquad \eta_6 = -M_2$

$\eta_7 = -M_1,\ \ \eta_8 = -W$

With the proof of Theorem 3.1 and Theorem 3.2, we can adequately confirm that the equilibrium point E_o is locally asymptotically stable if $R_0 < 1$, else it is unstable if $R_0 > 1$

3.4 Sensitivity Analysis of the Model Parameters

To determine the most influential parameters driving asthma transmission dynamics due to environmental pollution, a local sensitivity analysis was performed using the mechanistic reproduction number (R_0) derived in Section 3.2. The analysis quantifies how proportional changes in each model parameter affect R_0, allowing prioritization of intervention strategies.

Using the mechanistic form:

$R_0 = \gamma\ x\ P*\ x\ (C^1 + C_2\ + C_3)$, where $P* = \frac{q}{\eta\ + p}$ and pathway contributions:

$$C_1 = \frac{\lambda}{\lambda\ + \mu}\ \ x\ \frac{1}{r_1\ + \alpha_1 + \ \mu}$$

$$C_2 = \frac{\alpha_1}{\alpha_1\ + \mu}\ \ x\ \frac{1}{r_2\ + \alpha_2 + \ \mu}$$

$$C_3 = \frac{\alpha_2}{\alpha_2\ + \mu}\ \ x\ \frac{1}{r_3\ + \alpha_3 + \ \mu}$$

Finite-difference elasticity indices were computed for each parameter using baseline values from Table 3.1. The elasticity (S_i)of a parameter measures the proportional change in R_0 resulting from a 1% change in that parameter, defined as:

$$S_i = \frac{\delta R_0}{\delta p_i}\ \ x\ \frac{p_i}{R_0}$$

Table 3.1 summarizes the local sensitivity indices obtained from the analysis.

Parameter	Baseline Value	Elasticity (Sensitivity Index)
Q	5.0	+1.00
γ	0.05	+1.00

η	0.08	−0.92
μ	0.01067	−0.20
r_1	0.065	−0.12
r_2	0.065	−0.12
r_3	0.065	−0.08
α_1	0.02	−0.05
α_2	0.02	−0.04
p	0.01	−0.03

The results in Table 3.1 show that the aggregate emission rate of pollutants (q) and the environmental transmission coefficient (γ) have the highest positive elasticities, indicating that small increases in these parameters significantly raise R_0. In contrast, the pollutant attenuation rate (η) and recovery rates ($r_1 \ - r_s$) exhibit negative elasticities, showing that improving pollution removal or enhancing recovery interventions effectively reduce R_0. Thus, pollution control and behavioral interventions remain the most effective strategies for asthma mitigation. These findings corroborate the numerical results in Section 3.4 and reinforce the model's recommendation for policies targeting emission reduction, increased public awareness, and improved healthcare management.

3.5 Numerical Experiment of the Model

The Asthma Disease model due to environmental pollution in section 2.5, demonstrated in the model equation (2.1) - (2.8) was solved numerically using Runge-Kutta-Fehllberg 4-5th order method and implemented using Maple 17 Software (Maplesoft, Waterloo Maple Inc, 2017). The parameters used in the implementation of the model are shown in Table 3.1 below. Parameters were chosen in consonance with the threshold values obtained in the stability analysis of both the disease-free equilibrium and the endemic equilibrium state of the model.

Table 3.2 Estimated values of the parameters used in the Numerical experiments

Parameters	Values	Source		Parameters	Values	Source
$\boldsymbol{S(t)}$	48,714	Ozoh, *et al.* (2019)		α_1	0.05	Assumed
$\boldsymbol{E(t)}$	20,063	Ozoh, *et al.* (2019)		α_2	0.02	Assumed
$\boldsymbol{I_1(t)}$	8,530	Ozoh, *et al.* (2019)		α_3	0.02	Assumed
$\boldsymbol{I_2(t)}$	4,720	Ozoh, *et al.* (2019)		α_4	0.05	Assumed
$\boldsymbol{I_3(t)}$	3,560	Ozoh, *et al.* (2019)		σ_1	0.065	LHT (2025)
$\boldsymbol{I_4(t)}$	3,273	Ozoh, *et al.* (2019)		σ_2	0.065	LHT (2025)
$\boldsymbol{R(t)}$	2,560	Ozoh, *et al.* (2019)		σ_3	0.065	LHT (2025)
$\boldsymbol{P(t)}$	1000.3	Ram & Agraj (2009)		σ_4	0.065	LHT (2025)
$\boldsymbol{N(t)}$	92,420.3	NACA (2011)		σ_5	0.005	LHT (2025)
Λ	100	Ram & Agraj (2009)		ϖ	0.6	Assumed
$\boldsymbol{\gamma}$	0.0002	Olukayode *et al*, (2024)		τ	0.5	Assumed
$\boldsymbol{\lambda}$	0.0003	Olukayode *et al*, (2024)		δ	0.01	Junehyul, *et al* (2017)
$\boldsymbol{\beta_1}$	0.08	Wenjia Chen *et al.* (2018)		μ	0.01067	NACA (2011)
$\boldsymbol{\beta_2}$	0.08	Wenjia Chen *et al.* (2018)		Q	5 ton/year	Ram & Agraj (2009)
$\boldsymbol{\beta_3}$	0.08	Wenjia Chen *et al.* (2018)				

3.4.1 List of Numerical Experiments

The following numerical experiment were carried out

1) To determine the effect of varying transmission rate (γ) on the prevalence of asthmatic infection on the Exposed class $E(t)$ due to interaction of susceptible with environmental pollutants. $(\gamma = 0.002, 0.004, 0.006, 0.008)$
2) To determine the effect of varying transmission rate (λ) on prevalence of asthmatics infection on the Infectious class (I_1)due to interaction of Exposed individuals with environmental pollutants $(\lambda = 0.002, 0.004, 0.006, 0.008)$.
3) To determine the effect of successful asthma control due to public awareness (ϖ) on prevalence of asthmatics infection in the Infectious class (I_1) due to various strategies of interventions $(\varpi = 0.2, 0.4, 0.6, 0.8, 1.0)$.
4) To determine the effect of successful asthma control due to public awareness (ϖ) on recovered class $R(t)$ due to various probability of interventions $\varpi = (0.2, 0.4, 0.6, 0.8, 1.0)$
5) To determine the effect of varying Aggregate emission rate (Q) of pollutants on pollution over time $(Q = 5, 10, 15, 20, 25)$.
6) To determine the effect of Aggregate emission rate (Q) of pollutants on the susceptible population over time $(Q = 5, 10, 15, 20, 25)$
7) To determine the effect of Aggregate emission rate (Q) of pollutants on the Infected population over time $(Q = 10, 15, 20, 25, \lambda = 0.003, \gamma = 0.002)$

3.4.2 Graphical Representation of Results

Experiment 1: The effect of varying transmission rate (γ) on prevalence of asthmatics infection on the Exposed class due to interaction of susceptible with the environmental pollutant. $(\gamma = 0.002, 0.004, 0.006, 0.008)$

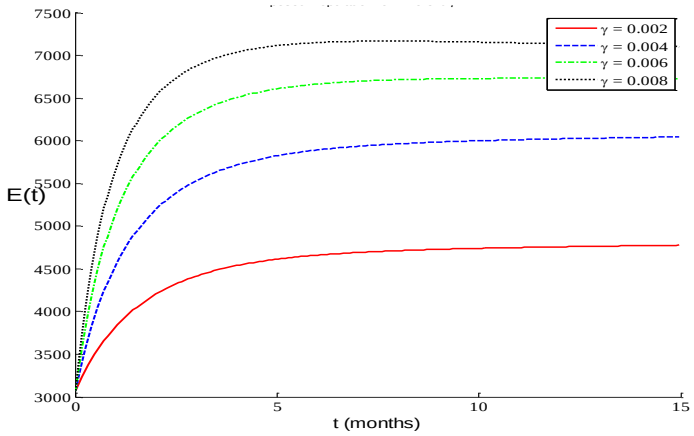

Figure 3.1 Prevalence of asthmatic infection in the exposed class with varying transmission rate of due to interaction of susceptible with environmental pollution. $(\gamma = 0.002, 0.004, 0.006, 0.008)$.

Experiment 2: The effect of varying transmission rate (λ) on prevalence of asthmatics infection in the Infectious class (I_1) due to interaction of Exposed individuals with environmental pollution $(\lambda = 0.002, 0.004, 0.006, 0.008)$

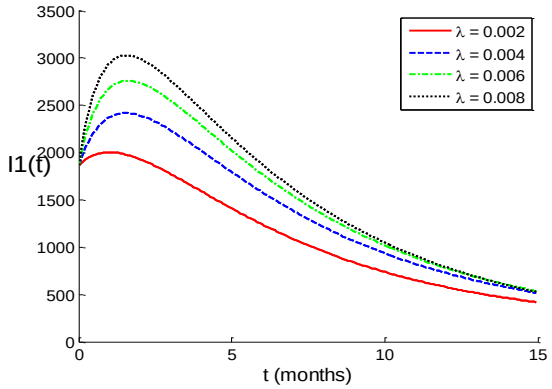

Figure 3.2 Prevalence of asthmatic infection in the Infectious class (I_1) - Mild Asthma with varying rate of interaction of exposed individuals with the environment. $(\gamma = 0.002, 0.004, 0.006, 0.008)$.

Experiment 3: The effect of successful asthma control due to public awareness (ϖ) on prevalence of asthmatics infection in the Infectious class (I_1) due to various strategies of interventions $(\boldsymbol{\varpi = 0.2, 0.4, 0.6, 0.8, 1.0})$

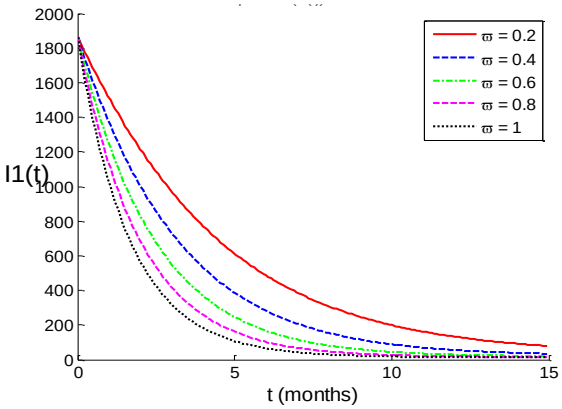

Figure 3.3 Prevalence of asthmatic infection in the Infectious class (I_1) - Mild Asthma with varying rate asthmatic control $(\varpi = 0.2, 0.4, 0.6, 0.8, 1.0)$.

Experiment 4: The effect of successful asthma control due to public awareness (ϖ) on recovered class $(R(t))$due to various probability of interventions $(\boldsymbol{\varpi = 0.2, 0.4, 0.6, 0.8, 1.0})$

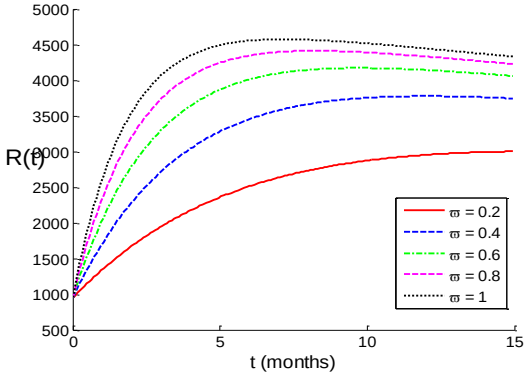

Figure 3.4 Impact of successful asthma control due to public awareness in the Recovered class (R) with varying control rate $(\varpi = 0.2, 0.4, 0.6, 0.8, 1.0)$.

Experiment 5: The effect of Aggregate emission rate (Q) of pollutants on pollution over time $(\boldsymbol{Q = 5, 10, 15, 20, 25})$

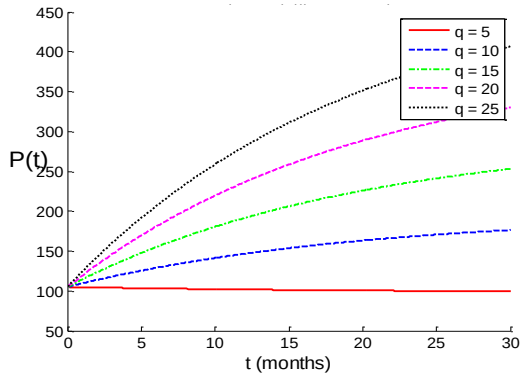

Figure 3.5 Impact of Cumulative density of pollutants on the pollution class (P) with varying aggregate emission rate (q) $(q = 5, 10, 15, 20, 25)$.

Experiment 6: The effect of Aggregate emission rate (Q) of pollutants on the susceptible population over time $(\boldsymbol{Q = 5, 10, 15, 20, 25})$

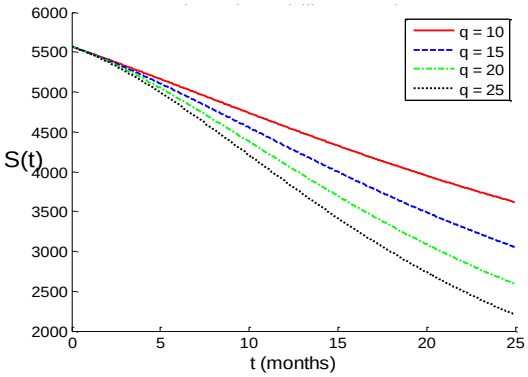

Figure 3.6 Impact of Cumulative density of pollutants on the Susceptible population class $(S(t))$ with varying aggregate emission rate (q) $(q = 10, 15, 20, 25)$.

Experiment 7: The effect of Aggregate emission rate (Q) of pollutants on the Infected population over time $(\boldsymbol{Q = 10, 15, 20, 25, \lambda = 0.003, \gamma = 0.002})$

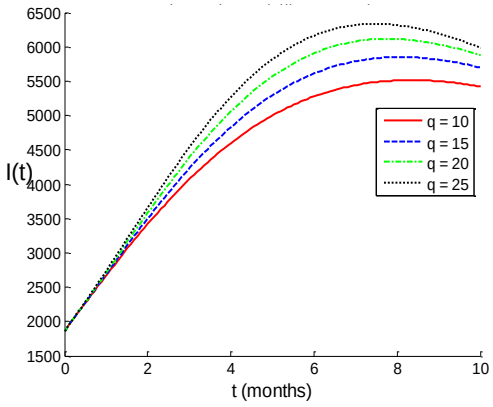

Figure 3.7 Impact of Cumulative density of pollutants on the Infected population class $(I(t))$ with varying aggregate emission rate (q) $(q = 10, 15, 20, 25)$.

DISCUSSION

This study analyzed the impact of environmental pollution on asthma dynamics using a compartmental mathematical model, integrating findings from numerical simulations with existing scholarly research to provide deeper insights into asthma transmission and control. The results demonstrated that as the environmental transmission coefficient (γ) increased, the number of exposed individuals rose exponentially, underscoring the significant role of pollution in asthma progression. This finding aligns with the work of Naresh and Tripathi (2009), who linked pollutant concentrations to increased asthma prevalence, and with the WHO (2016) report attributing 43% of global asthma cases to air pollution [1]. Thus, γ serves as a crucial determinant of exposure levels, emphasizing the importance of pollution reduction in mitigating asthma risks.

Furthermore, the study revealed that higher exposure transmission rates (λ) substantially increased all infected classes, ranging from mild to chronic asthma. This supports the findings of Olukayode et al. (2024) and other empirical studies (Chen et al., 2013; Liu et al., 2018), which showed that prolonged exposure to pollutants accelerates disease manifestation and hospital admissions. By dividing infected individuals into multiple severity stages, this model enhances precision and captures the heterogeneous nature of asthma progression more effectively than previous models. [2]

The model also examined the influence of public health awareness and behavioral change (ϖ), demonstrating that increased awareness leads to a significant decline in infection rates and a corresponding rise in recovery levels. This mirrors the results of Bateman et al. (2018) and Zhang and Jiang (2014), who observed notable improvements in asthma control following targeted awareness campaigns. [3] The inclusion of ϖ as a behavioral control parameter represents an important innovation, as it quantifies the impact of soft interventions—such as education and preventive behaviors—on disease dynamics, highlighting the need for continuous community outreach and education.

Additionally, the pollutant emission rate (q) was shown to drive an increase in environmental load ($P(t)$), which in turn decreased the susceptible population and increased both exposure and infection rates. This outcome aligns with the findings of Naresh and Tripathi (2009) and the Global Burden of Disease (2019) report, which identified fine particulate matter as a leading cause of asthma morbidity. [4] The model thereby quantifies the feedback loop between pollutant emissions and asthma prevalence, emphasizing that controlling emission rates is vital for reducing environmental health risks. Long-term analysis further revealed that under conditions of high pollution and weak intervention (low ϖ), the infected population fails to reach a disease-free equilibrium, instead stabilizing at an endemic state. This aligns with global observations in highly industrialized regions such as Nigeria, India, and China (WHO, 2021; Adewale et al., 2023), where chronic asthma remains persistent without sustained intervention. [5] Overall, this study contributes significantly to existing literature by extending earlier models through the integration of multiple infection stages, recovery dynamics, and pollution feedback mechanisms. By introducing control parameters such as ϖ, q, γ, and λ, the model establishes a direct link between public health actions, pollution control policies, and asthma outcomes. These innovations enhance the model's realism and provide valuable guidance for designing effective intervention strategies, particularly in pollution-prone and resource-limited settings.

CONCLUSION

In conclusion, this research reinforces the well-established but often underappreciated link between environmental pollution and asthma dynamics. Through the use of a robust mathematical framework and simulations, it was demonstrated that pollution not only increases the rate of asthma incidence but also makes eradication more difficult unless strong and sustained

interventions are adopted. The disease-free equilibrium is only attainable when key parameters, such as pollutant load (q), exposure rates $(\gamma, \lambda)$are reduced and and public awareness (ϖ)enhanced to maintain thresholds that bring the basic reproduction number and public awareness (R_0)below unity. The model emphasizes that asthma is not only a biomedical condition but also a socio-environmental one that requires interdisciplinary strategies for control and prevention. This study validates existing literature while offering new contributions, especially the inclusion of behavior-based intervention strategies and feedback loops involving environmental pollutants. It underlines the potential of mathematical modeling in health forecasting and the formulation of data-driven policies in environmental and respiratory health.

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