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Modelling asthma development in a population with genetic risk and polluted environment

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Abstract. Environmental pollutant continues to pose a great threat to public health, leading to development of chronic diseases. In this study, a nonlinear mathematical model is formulated and analysed to study the effect of genetic risk, environmental pollutant, public health education/awareness on asthma development. Conditions for the existence of the unique positive steady state and permanence of the system are assessed. Using Lyapunov function analysis, the unique positive steady state is locally and globally asymptotically stable. Results reveal that genetic risk, pollutant emission rate, effective exposure rate of population to polluted environment and recurrence rate contribute to asthma prevalence. However, sufficiently effective pollutant reduction strategies, improvement in compliance to public health education/awareness together with human dependent environmental pollutant depletion lead to a marked reduction in disease prevalence.

Keywords: mathematical modelling, asthma, environmental pollutant, family history, awareness.

1 Introduction

Asthma is a chronic inflammatory airway disease characterised by reversible airway obstruction and airway hyper-responsiveness [3] often in response to triggers such as allergens, pollutant released into the environment or viral infections. Development of asthma may be due to either environmental exposures in the absence of genetic predisposition [5] or a combination of multiple genes (family history) and environmental factors [21]. However, having a family history with environmental pollutant, may increase a person's risk of developing asthma [19].

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Environmental pollutant emitted from different sources (i.e. industries, vehicles, chemicals, cigarette smoke, pollen, deforestation etc.) are partly as a result of human activities and continues to worsen with *particulate matter* $(\mathrm{PM})_{2.5}$ (particles that have diameter less than 2.5 micrometres) concentration, ranging from $99.3\ \mu\mathrm{g/m}^3$ to $152.6\ \mu\mathrm{g/m}^3$ in the case of Uganda [11]. Environmental pollutant contributes to the development of a number of human diseases e.g. respiratory diseases (tuberculosis, chronic obstructive pulmonary disease (COPD)), heart diseases, lung cancer, asthma etc. If environmental pollutant is controlled, disease development can be reduced. Also, asthma awareness/education has been suggested as one way to improve on asthma control, and if asthma is properly treated, control can be achieved, and a person lives a normal active life [4, 10, 18, 20].

A number of nonlinear mathematical models have been proposed and analysed to study the environment with biological species, the effect of environmental pollutant to biological species and in particular to the development of diseases [1, 7, 9, 13, 16, 17]. Agarwal and Devi [1] studied the effect of environmental tax on the survival of biological species affected by pollutant in the environment and showed that the density of the biological species may become extinct with increasing rate of emission of pollutant in the environment without tax. However, with imposition of environmental tax, the density of biological species can be maintained at a desired level. Ghosh [8] studied the effect of inhaled pollutant on the development of asthma in a population with constant immigration and logistic growth. In each case, release of pollutant into the environment was considered to be either constant, population dependent or periodic. It was shown that increase in pollutant led to increase in asthmatic (diseased) population in the region under consideration.

In the above studies, the influence of genetic risk with environmental pollutant on the development of asthma as well as the control of asthma through treatment, provision of public health awareness and population dependent depletion of environmental pollutant have not been considered. Therefore, formulation and analysis of a mathematical model is done to get a clear understanding of how different levels of environmental pollutant together with family history influence the development of asthma; incorporating compliance to public health awareness as a control measure of asthma on top of treatment.

2 Materials and methods

In this section, a mathematical model incorporating factors that promote the development and control of asthma is presented. It is assumed that the human population is affected by pollutant in the environment. Increase in asthma disease is caused by a combination of individual's genetic make up and environmental pollutant. Family history is accounted for by considering two classes of susceptible subpopulation i.e. susceptible with family history (high genetic risk) and susceptible without family history (low genetic risk). A proportion of the recruitment into the human population have family history of asthma or allergies. Those with family history of asthma or allergies are more likely to develop asthma than those with no family history [2].

It is also assumed that all susceptible population are simultaneously affected by environmental exposure, which is contributed to through indoor and outdoor pollutant.

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Development of asthma may occur to individuals with high or low genetic risk if there is adequate exposure to environmental pollutant i.e. after a single exposure to relatively high doses of environmental pollutant or after continuous exposure to environmental pollutant.

It is considered that individuals with asthma can gain control of asthma symptoms through adherence to treatment, and control may be improved through public health education/awareness of asthma or avoidance of environment triggers (pollutant). The controlled asthmatic may experience recurring or worsening of asthma symptoms through re-exposure to environmental pollutant.

In addition, the concentration of environmental pollutant is assumed to decrease due to some natural degradation and depletion initiated by human population effort.

In model (1)–(5), the human population density $N(t)$ is divided into four classes: two susceptible classes depending on their family history (the susceptible class with family history of asthma or allergies ($T(t)$) and the susceptible class without family history of asthma or allergies ($S(t)$)), the asthmatic class ($A(t)$) and the controlled asthmatic class ($C(t)$). $E(t)$ is the cumulative concentration of pollutant (indoor and outdoor pollutant) present in the environment under consideration.

Λ is the per capita recruitment rate into the human population. ρ is the proportion of human recruitment that have family history of asthma or allergies. β is the effective exposure rate of susceptible individuals to polluted environment leading to development of asthma. γ is the modification parameter that accounts for assumed reduced development rate of asthma among susceptible individuals without family history. α is the average rate of asthma control, ω is the probability of success of asthma control through public health education/awareness. ν is the recurring rate of asthma among the controlled asthmatic. μ and σ are the natural and disease related death rate, respectively. Q is the cumulative rate of emission of pollutant into the environment from various sources, δ is the natural depletion rate coefficient of pollutant, τ is the depletion rate coefficient of the pollutant in the environment by human population activities.

Putting all the definitions and assumptions together, the model for the dynamics of asthma development is given by the following system of nonlinear ordinary differential equations:

$$\frac{\mathrm{d}T}{\mathrm{d}t} = \rho\Lambda - \beta TE - \mu T, \tag{1}$$

$$\frac{\mathrm{d}S}{\mathrm{d}t} = (1-\rho)\Lambda - \gamma\beta SE - \mu S, \tag{2}$$

$$\frac{\mathrm{d}A}{\mathrm{d}t} = \beta TE + \gamma\beta SE + \nu CE - (\omega\alpha + \sigma + \mu)A, \tag{3}$$

$$\frac{\mathrm{d}C}{\mathrm{d}t} = \omega\alpha A - (\nu E + \mu)C, \tag{4}$$

$$\frac{\mathrm{d}E}{\mathrm{d}t} = Q - (\delta + \tau N)E. \tag{5}$$

Adding up the equations for $T(t)$, $S(t)$, $A(t)$ and $C(t)$, we obtain

$$\frac{\mathrm{d}N(t)}{\mathrm{d}t} = \Lambda - \sigma A - \mu N(t)$$

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with $T(0) = T_0 \geqslant 0$, $S(0) = S_0 \geqslant 0$, $A(0) = A_0 \geqslant 0$, $C(0) = C_0 \geqslant 0$, $E(0) = E_0 \geqslant 0$ and $N(0) = N_0 \geqslant 0$, $0 < \rho < 1$, $0 < \gamma < 1$, $0 < \omega < 1$. Assuming that all variables and parameters of the system of equations in (1)–(5) are non-negative for all $t \geqslant 0$.

3 Model analysis

3.1 Positivity and boundedness of the solution

In order to analyse system (1)–(5), we find the bounds of all dependent variables involved in the model. It is enough to show that the solution of system (1)–(5) is bounded in the proper subset $\Omega \subset \mathbb{R}^4_+ \times \mathbb{R}_+$ with $\Omega = \Omega_h \times \Omega_e$ for all $t \geqslant 0$, the region of attraction for all solutions of system (1)–(5) initiating in the interior of the positive orthant, where

$$\Omega_h = \left\{(T, S, A, C) \in \mathbb{R}^4_+ \colon 0 \leqslant T(t) + S(t) + A(t) + C(t) \leqslant N(t) \leqslant \frac{\Lambda}{\mu}\right\}$$

and

$$\Omega_e = \left\{E \in \mathbb{R}_+ \colon 0 \leqslant E(t) \leqslant \frac{Q}{\delta}\right\}.$$

The assumption is that all initial values of the dependent variables considered in system (1)–(5) belongs to the region Ω and are positive. Using equations (1)–(4) gives $\mathrm{d}N(t)/\mathrm{d}t \leqslant \Lambda - \mu N(t)$. Solving for $N(t)$, we obtain that $N(t) \leqslant \Lambda/\mu + (N_0 - \Lambda/\mu)\mathrm{e}^{\mu t}$ implying that $\lim_{t\to\infty} \sup N(t) \leqslant \Lambda/\mu$. Thus, the feasible solution set of the human population in system (1)–(5) is contained in the region

$$\Omega_h = \left\{(T, S, A, C) \in \mathbb{R}^4_+ \colon 0 \leqslant N(t) \leqslant \frac{\Lambda}{\mu}\right\}.$$

Similarly, considering environmental exposure, $E(t)$, and using the differential equation (5), we note that $\lim_{t\to\infty} \sup E \leqslant Q/\delta$.

Hence, the feasible solution set of environmental exposure in system (1)–(5) is contained in

$$\Omega_e = \left\{E \in \mathbb{R}_+ \colon 0 \leqslant E(t) \leqslant \frac{Q}{\delta}\right\}.$$

Therefore, the solution set of system (1)–(5) is bounded in $\Omega = \Omega_h \times \Omega_e$ in which it is biologically and mathematically well-posed.

In addition, the region $\Omega \subset \mathbb{R}^5_+$ is positively invariant in relation to system (1)–(5), and a non-negative solution exists for all $0 < t < \infty$.

Suppose that the initial state variables are non-negative, it can be shown that solutions of system (1)–(5) remain positive for all $t > 0$.

Assume that for Eq. (1), on the contrary, there exists a time τ such that $T(\tau) = 0$, $\mathrm{d}T/\mathrm{d}\tau \leqslant 0$ with $S(t), A(t), C(t), E(t) > 0$ for $0 < t < \tau$. Then [14]

$$\frac{\mathrm{d}T(\tau)}{\mathrm{d}\tau} = \rho\Lambda - \beta T(\tau)E(\tau) - \mu T(\tau) = \rho\Lambda > 0 \quad \text{for } T(\tau) = 0,$$

which is a contradiction. Thus, $T(t) > 0$ for all $0 < t < \tau$.

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In the same way, it can be shown that variables $S(t)$, $A(t)$, $C(t)$ and $E(t)$ remain positive for all $t > 0$.

3.2 Equilibrium analysis

System (1)–(5) has a unique positive equilibrium point $x(\widetilde{T}, \widetilde{S}, \widetilde{A}, \widetilde{C}, \widetilde{E})$ in T, S, A, C, E-space.

3.2.1 Existence of $x(\widetilde{T}, \widetilde{S}, \widetilde{A}, \widetilde{C}, \widetilde{E})$

Setting $\mathrm{d}T/\mathrm{d}t$, $\mathrm{d}S/\mathrm{d}t$ and $\mathrm{d}C/\mathrm{d}t$ from system (1)–(5) to zero gives the following equations:

$$T(t) = \frac{\rho\Lambda}{\beta E + \mu} = f_1(E), \qquad S(t) = \frac{(1-\rho)\Lambda)}{\gamma\beta E + \mu} = f_2(E), \tag{6}$$

$$C(t) = \frac{\omega\alpha A}{\nu E + \mu}. \tag{7}$$

Equations (6) and (7) together with $\mathrm{d}A/\mathrm{d}t = 0$ give

$$A(t) = \frac{\beta E(\nu E + \mu)[f_1(E) + \gamma f_2(E)]}{\mu(\omega\alpha + \sigma + \mu) + \nu E(\sigma + \mu)} = f_3(E), \tag{8}$$

$$C(t) = \frac{\omega\alpha\beta E[f_1(E) + \gamma f_2(E)]}{\mu(\omega\alpha + \sigma + \mu) + \nu E(\sigma + \mu)} = f_4(E). \tag{9}$$

To show the existence of equilibrium point x, we define a function $F(E) = Q - (\delta + \tau N)E$ from Eq. (5). Using $N(t) = T(t) + S(t) + A(t) + C(t)$ with Eqs. (6), (8) and (9) in $F(E)$ gives a function of E

$$F(E) = Q - \delta E - \tau E\left[f_1(E) + f_2(E) + f_3(E) + f_4(E)\right]. \tag{10}$$

It is noted from Eq. (10) that $F(0) = Q > 0$ and

$$F\left(\frac{Q}{\delta}\right) = -\frac{Q}{\delta}\tau\left[f_1\left(\frac{Q}{\delta}\right) + f_2\left(\frac{Q}{\delta}\right) + f_3\left(\frac{Q}{\delta}\right) + f_4\left(\frac{Q}{\delta}\right)\right] < 0,$$

where

$$f_1\left(\frac{Q}{\delta}\right) = \frac{\rho\Lambda}{\beta(\frac{Q}{\delta}) + \mu} > 0, \quad f_2\left(\frac{Q}{\delta}\right) = \frac{(1-\rho)\Lambda}{\gamma\beta(\frac{Q}{\delta}) + \mu} > 0 \quad \text{for } 0 < \rho < 1,$$

$$f_3\left(\frac{Q}{\delta}\right) = \frac{\beta(\frac{Q}{\delta})(\nu(\frac{Q}{\delta}) + \mu)[f_1(\frac{Q}{\delta}) + \gamma f_2(\frac{Q}{\delta})]}{\mu(\omega\alpha + \sigma + \mu) + \nu(\frac{Q}{\delta})(\sigma + \mu)} > 0$$

and

$$f_4\left(\frac{Q}{\delta}\right) = \frac{\omega\alpha\beta(\frac{Q}{\delta})[f_1(\frac{Q}{\delta}) + \gamma f_2(\frac{Q}{\delta})]}{\mu(\omega\alpha + \sigma + \mu) + \nu(\frac{Q}{\delta})(\sigma + \mu)} > 0.$$

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This implies that $F(\widetilde{E}) = 0$ will have a root $\widetilde{E}$ in the interval $0 < \widetilde{E} < Q/\delta$ since $F(0) > 0$ and $F(Q/\delta) < 0$.

Similarly, expressions of $\widetilde{T}$, $\widetilde{S}$, $\widetilde{A}$ and $\widetilde{C}$ can be obtained from (6), (8) and (9), respectively, to show existence of a root.

3.2.2 *Uniqueness of* $x(\widetilde{T}, \widetilde{S}, \widetilde{A}, \widetilde{C}, \widetilde{E})$

The condition for x to be unique is $\mathrm{d}F/\mathrm{d}E < 0$ at the equilibrium point x. Given

$$\begin{aligned} F(E) &= Q - \delta E - \tau E\big[f_1(E) + f_2(E) + f_3(E) + f_4(E)\big], \\ \frac{\mathrm{d}F}{\mathrm{d}E} &= -\delta - \tau\big[f_1(E) + f_2(E) + f_3(E) + f_4(E)\big] \\ &\quad - \tau E\big[f_1'(E) + f_2'(E) + f_3'(E) + f_4'(E)\big] \end{aligned}$$

with

$$\begin{aligned} f_1'(E) &= \frac{-\rho\Lambda\beta}{(\beta E + \mu)^2}, \qquad f_2'(E) = \frac{(\rho - 1)\Lambda\gamma\beta}{(\gamma\beta E + \mu)^2}, \\ f_3'(E) &= \frac{\beta\big[\mu(2\nu E + \mu)(\omega\alpha + \sigma + \mu) + (\nu E)^2(\sigma + \mu)\big](f_1(E) + \gamma f_2(E))}{(\mu(\omega\alpha + \sigma + \mu) + \nu E(\sigma + \mu))^2} \\ &\quad + \frac{\beta E(\nu E + \mu)(f_1'(E) + \gamma f_2'(E))}{(\mu(\omega\alpha + \sigma + \mu) + \nu E(\sigma + \mu))} \end{aligned}$$

and

$$\begin{aligned} f_4'(E) &= \frac{\omega\alpha\beta\mu(\omega\alpha + \sigma + \mu)(f_1(E) + \gamma f_2(E))}{(\mu(\omega\alpha + \sigma + \mu) + \nu E(\sigma + \mu))^2} \\ &\quad + \frac{\omega\alpha\beta E(f_1'(E) + \gamma f_2'(E))}{(\mu(\omega\alpha + \sigma + \mu) + \nu E(\sigma + \mu))}. \end{aligned}$$

Then

$$\begin{aligned} \frac{\mathrm{d}F}{\mathrm{d}E} &= -\delta - \tau\big[\big(f_1(E) + Ef_1'(E)\big) + \big(f_2(E) + Ef_2'(E)\big) \\ &\quad + \big(f_3(E) + Ef_3'(E)\big) + \big(f_4(E) + Ef_4'(E)\big)\big] < 0, \end{aligned}$$

implying that equilibrium point x is unique.

3.3 Stability analysis of $x(\widetilde{T}, \widetilde{S}, \widetilde{A}, \widetilde{C}, \widetilde{E})$

3.3.1 *Local stability*

Stability behaviour of equilibrium point x can be studied by using the method of Lyapunov's function. The following theorem gives the conditions under which equilibrium point x is locally asymptotically stable.

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Theorem 1. *Let the following inequality holds*:

$$\max\left\{\frac{(6\tau\widetilde{E})^3}{(\delta+\tau\widetilde{N})^3}, \frac{27\gamma^2(\beta\widetilde{E})^4(\nu^2\widetilde{E}\widetilde{C}+\omega\alpha(\beta\widetilde{T}+\gamma\beta\widetilde{S}+\nu\widetilde{C}))^2}{8(\nu\widetilde{C})^2(\omega\alpha+\sigma+\mu)^3(\beta\widetilde{T}+\gamma\beta\widetilde{S}+\nu\widetilde{C})^3}\right\} < \frac{(\beta\widetilde{E}+\mu)(\gamma\beta\widetilde{E}+\mu)(\nu\widetilde{E}+\mu)}{(\beta\widetilde{T})(\gamma\beta\widetilde{S})(\nu\widetilde{C})}. \tag{11}$$

Then x is locally asymptotically stable.

Proof. Linearisation of system (1)–(5) about the positive equilibrium x is carried out first by taking the following transformations: $T = \widetilde{T} + t_1$, $S = \widetilde{S} + s_1$, $A = \widetilde{A} + a_1$, $C = \widetilde{C} + c_1$, $E = \widetilde{E} + e_1$, where t_1, s_1, a_1, c_1 and e_1 are small perturbations about x. Considering the following positive definite function in the linearised system:

$$V_1 = \frac{1}{2}k_1t_1^2 + \frac{1}{2}k_2s_1^2 + \frac{1}{2}k_3a_1^2 + \frac{1}{2}k_4c_1^2 + \frac{1}{2}e_1^2,$$

where k_1, k_2, k_3, k_4 are some positive constant to be determined appropriately. Computing derivative of V_1 with respect to time t, $\mathrm{d}V_1/\mathrm{d}t$ can be found along the solutions of the linearised system as follows:

$$\begin{aligned}\frac{\mathrm{d}V_1}{\mathrm{d}t} &= \left[-k_1(\beta\widetilde{E}+\mu)t_1^2 - k_2(\gamma\beta\widetilde{E}+\mu)s_1^2 - k_3(\omega\alpha+\sigma+\mu)a_1^2\right]\\ &\quad+\left[-k_4(\nu\widetilde{E}+\mu)c_1^2 - (\delta+\tau\widetilde{N})e_1^2\right]\\ &\quad+\left[-(k_4\nu\widetilde{C}+\tau\widetilde{E})e_1c_1 + k_3((\beta\widetilde{T}+\gamma\beta\widetilde{S}+\nu\widetilde{C})-\tau\widetilde{E})a_1e_1\right]\\ &\quad+\left[-(k_1\beta\widetilde{T}+\tau\widetilde{E})e_1t_1 - (k_2\gamma\beta\widetilde{S}+\tau\widetilde{E})e_1s_1\right]\\ &\quad+\left[+(k_3\nu\widetilde{E}+k_4\omega\alpha)a_1c_1 + k_3\beta\widetilde{E}a_1t_1 + k_3\gamma\beta\widetilde{E}a_1s_1\right].\end{aligned}$$

Choosing

$$k_1 = \frac{\tau\widetilde{E}}{\beta\widetilde{T}}, \quad k_2 = \frac{\tau\widetilde{E}}{\gamma\beta\widetilde{S}}, \quad k_3 = \frac{\tau\widetilde{E}}{(\beta\widetilde{T}+\gamma\beta\widetilde{S}+\nu\widetilde{C})} \quad \text{and} \quad k_4 = \frac{\tau\widetilde{E}}{\nu\widetilde{C}},$$

the sufficient conditions for $\mathrm{d}V_1/\mathrm{d}t$ to be negative definite are that the following inequalities hold:

$$6\tau\widetilde{E} < \frac{(\beta\widetilde{E}+\mu)(\delta+\tau\widetilde{N})}{\beta\widetilde{T}}, \qquad 6\tau\widetilde{E} < \frac{(\gamma\beta\widetilde{E}+\mu)(\delta+\tau\widetilde{N})}{\gamma\beta\widetilde{S}},$$

$$6\tau\widetilde{E} < \frac{(\nu\widetilde{E}+\mu)(\delta+\tau\widetilde{N})}{\nu\widetilde{C}}, \qquad \frac{(\beta\widetilde{E})^2}{(\beta\widetilde{T}+\gamma\beta\widetilde{S}+\nu\widetilde{C})} < \frac{2}{3(\beta\widetilde{T})}(\beta\widetilde{E}+\mu)(\omega\alpha+\sigma+\mu),$$

$$\frac{(\gamma\beta\widetilde{E})^2}{(\beta\widetilde{T}+\gamma\beta\widetilde{S}+\nu\widetilde{C})} < \frac{2}{3(\gamma\beta\widetilde{S})}(\gamma\beta\widetilde{E}+\mu)(\omega\alpha+\sigma+\mu),$$

$$\frac{(\nu^2\widetilde{E}\widetilde{C}+\omega\alpha(\beta\widetilde{T}+\gamma\beta\widetilde{S}+\nu\widetilde{C}))^2}{(\beta\widetilde{T}+\gamma\beta\widetilde{S}+\nu\widetilde{C})\nu\widetilde{C}} < \frac{2}{3}(\nu\widetilde{E}+\mu)(\omega\alpha+\sigma+\mu).$$

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Putting together the above inequalities, $\mathrm{d}V_1/\mathrm{d}t$ will be negative definite under inequality (11) showing that V_1 is a Lyapunov's function. Hence, the equilibrium point x is locally asymptotically stable under inequality (11). □

3.3.2 *Global stability*

Theorem 2. *Let the following inequality holds in* Ω:

$$\max\left\{\frac{(6\tau\widetilde{E})^3}{(\delta+\tau\widetilde{N})^3}, \frac{27\gamma^2(\beta\widetilde{E})^4(\nu^2\widetilde{E}\widetilde{C}+\omega\alpha(\beta\widetilde{T}+\gamma\beta\widetilde{S}+\nu\widetilde{C}))^2}{8(\nu\widetilde{C})^2(\omega\alpha+\sigma+\mu)^3(\beta\widetilde{T}+\gamma\beta\widetilde{S}+\nu\widetilde{C})^3}\right\} < \frac{(\beta\widetilde{E}+\mu)(\gamma\beta\widetilde{E}+\mu)(\nu\widetilde{E}+\mu)}{(\beta\widetilde{T})(\gamma\beta\widetilde{S})(\nu\widetilde{C})}. \tag{12}$$

Then x *is globally asymptotically stable.*

Proof. Consider the following Lyapunov function about equilibrium point x:

$$V_2 = \frac{c_1}{2}(T-\widetilde{T})^2 + \frac{c_2}{2}(S-\widetilde{S})^2 + \frac{c_3}{2}(A-\widetilde{A})^2 + \frac{c_4}{2}(C-\widetilde{C})^2 + \frac{1}{2}(E-\widetilde{E})^2,$$

where c_1, c_2, c_3, c_4 are some constant to be chosen appropriately. It can easily be shown that the function V_2 is zero at equilibrium point x and positive for all other positive values of T, S, A, C and E. Differentiating V_2 with respect to time t and substituting values of $\mathrm{d}T/\mathrm{d}t$, $\mathrm{d}S/\mathrm{d}t$, $\mathrm{d}A/\mathrm{d}t$, $\mathrm{d}C/\mathrm{d}t$ and $\mathrm{d}E/\mathrm{d}t$ from system (1)–(5) in $\mathrm{d}V_2/\mathrm{d}t$ give

$$\begin{aligned}\frac{\mathrm{d}V_2}{\mathrm{d}t} &= \left[-c_1(\beta\widetilde{E}+\mu)(T-\widetilde{T})^2 - c_2(\gamma\beta\widetilde{E}+\mu)(S-\widetilde{S})^2\right]\\ &+ \left[-c_3(\omega\alpha+\sigma+\mu)(A-\widetilde{A})^2 - c_4(\nu\widetilde{E}+\mu)(C-\widetilde{C})^2 - (\delta+\tau\widetilde{N})(E-\widetilde{E})^2\right]\\ &+ \left[-(c_4\nu\widetilde{C}+\tau\widetilde{E})(E-\widetilde{E})(C-\widetilde{C}) - (c_1\beta\widetilde{T}+\tau\widetilde{E})(E-\widetilde{E})(T-\widetilde{T})\right]\\ &+ \left[+c_3((\beta\widetilde{T}+\gamma\beta\widetilde{S}+\nu\widetilde{C})-\tau\widetilde{E})(E-\widetilde{E})(A-\widetilde{A})\right]\\ &+ \left[-(c_2\gamma\beta\widetilde{S}+\tau\widetilde{E})(E-\widetilde{E})(S-\widetilde{S}) + c_3\beta\widetilde{E}(T-\widetilde{T})(A-\widetilde{A})\right]\\ &+ \left[+(c_3\nu\widetilde{E}+c_4\omega\alpha)(C-\widetilde{C})(A-\widetilde{A}) + c_3\gamma\beta\widetilde{E}(S-\widetilde{S})(A-\widetilde{A})\right].\end{aligned}$$

Choosing

$$c_1 = \frac{\tau\widetilde{E}}{\beta\widetilde{T}}, \quad c_2 = \frac{\tau\widetilde{E}}{\gamma\beta\widetilde{S}}, \quad c_3 = \frac{\tau\widetilde{E}}{(\beta\widetilde{T}+\gamma\beta\widetilde{S}+\nu\widetilde{C})} \quad \text{and} \quad c_4 = \frac{\tau\widetilde{E}}{\nu\widetilde{C}},$$

the sufficient conditions for $\mathrm{d}V_2/\mathrm{d}t$ to be negative definite inside Ω, the region of attraction, are that the following inequalities hold:

$$6\tau\widetilde{E} < \frac{(\beta\widetilde{E}+\mu)(\delta+\tau\widetilde{N})}{\beta\widetilde{T}}, \qquad 6\tau\widetilde{E} < \frac{(\gamma\beta\widetilde{E}+\mu)(\delta+\tau\widetilde{N})}{\gamma\beta\widetilde{S}},$$

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$$6\tau\widetilde{E} < \frac{(\nu\widetilde{E}+\mu)(\delta+\tau\widetilde{N})}{\nu\widetilde{C}},$$

$$\frac{(\beta\widetilde{E})^2}{(\beta\widetilde{T}+\gamma\beta\widetilde{S}+\nu\widetilde{C})} < \frac{2}{3(\beta\widetilde{T})}(\beta\widetilde{E}+\mu)(\omega\alpha+\sigma+\mu),$$

$$\frac{(\gamma\beta\widetilde{E})^2}{(\beta\widetilde{T}+\gamma\beta\widetilde{S}+\nu\widetilde{C})} < \frac{2}{3(\gamma\beta\widetilde{S})}(\gamma\beta\widetilde{E}+\mu)(\omega\alpha+\sigma+\mu),$$

$$\frac{(\nu^2\widetilde{E}\widetilde{C}+\omega\alpha(\beta\widetilde{T}+\gamma\beta\widetilde{S}+\nu\widetilde{C}))^2}{(\beta\widetilde{T}+\gamma\beta\widetilde{S}+\nu\widetilde{C})\nu\widetilde{C}} < \frac{2}{3}(\nu\widetilde{E}+\mu)(\omega\alpha+\sigma+\mu).$$

Putting together the above inequalities, $\mathrm{d}V_2/\mathrm{d}t$ will be negative definite under the inequality (12) showing that V_2 is a Lyapunov's function. Hence, the equilibrium point x is globally asymptotically stable under inequality (12). □

Remark 1. It is observed from Theorems 1 and 2 that the unique positive equilibrium state is locally and globally asymptotically stable for small values of β, γ and ν, and stability conditions (11) and (12) will be satisfied. Implying that large values of the effective exposure rate of susceptible individuals to polluted environment, the assumed reduced development of asthma of the susceptible individuals with low genetic risk and recurrence rate of asthma, respectively, destabilises the system.

Remark 2. Also, the unique positive equilibrium state is locally and globally asymptotically stable for large values of α, ω, δ and τ, and stability conditions (11) and (12) will be satisfied; implying that small values of the rate of control of asthma, probability of success of asthma control through public health education/awareness, natural depletion rate of pollutant and depletion rate coefficient of the pollutant concentration in the environment by human population, respectively, destabilise the system.

In other words, stability analysis of the unique positive equilibrium state shows that an increase in the effective exposure rate to polluted environment and recurrence results in an increase of disease prevalence. However, adherence/compliance to public health education/awareness of asthma and human dependent depletion of environmental pollutant leads to low asthma development and recurrence, and then more controlled asthma is achieved.

3.4 Permanence of solutions

Permanence shows that with the initial presence of the population in the system, the population will survive in the future time. Mathematically, the population $V(t)$ is said to persist (strongly persist) if $V(0) > 0$ implies $V(t) > 0$ and $\lim_{t\to\infty} \inf V(t) > 0$. Also, system (1)–(5) is said to be uniformly persistent (permanence) if for each $i = 1, 2, \ldots, n$, there exists positive constants m_i and M_i such that $0 < m_i \leqslant \lim_{t\to\infty} \inf V_i(t) \leqslant \lim_{t\to\infty} \sup V_i(t) \leqslant M_i$, where $V_i(t)$ denotes the positive solution of system (1)–(5) with corresponding positive initial values [6, 13]. Finally, we say that a system uniformly persists whenever each component uniformly persists.

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Theorem 3. *System* (1)–(5) *is uniformly persistent if the proportion of human recruitment with family history* $\rho < 1$.

Proof. Considering Eq. (1), we have

$$\frac{\mathrm{d}T}{\mathrm{d}t} \geqslant \rho\Lambda - \left(\beta\frac{Q}{\delta} + \mu\right)T$$

thus,

$$\lim_{t\to\infty} \inf T(t) \geqslant \frac{\rho\Lambda}{\beta\frac{Q}{\delta} + \mu} = T_{\min}.$$

Also, from Eq. (2) we can write

$$\frac{\mathrm{d}S}{\mathrm{d}t} \geqslant (1-\rho)\Lambda - \left(\gamma\beta\frac{Q}{\delta} + \mu\right)S,$$

implying that

$$\lim_{t\to\infty} \inf S(t) \geqslant \frac{(1-\rho)\Lambda}{\gamma\beta\frac{Q}{\delta} + \mu} = S_{\min},$$

here S_{min} is positive, provided that $\rho < 1$. Again, from Eq. (3) we have

$$\frac{\mathrm{d}A}{\mathrm{d}t} \geqslant \beta\frac{Q}{\delta}(T_{\min} + \gamma S_{\min}) - (\omega\alpha + \sigma + \mu)A,$$

giving

$$\lim_{t\to\infty} \inf A(t) \geqslant \frac{\beta\frac{Q}{\delta}(T_{\min} + \gamma S_{\min})}{\omega\alpha + \sigma + \mu} = A_{\min}.$$

Further, from Eq. (4) we have

$$\frac{\mathrm{d}C}{\mathrm{d}t} \geqslant \omega\alpha A_{\min} - \left(\nu\frac{Q}{\delta} + \mu\right)C,$$

it follows that

$$\lim_{t\to\infty} \inf C(t) \geqslant \frac{\omega\alpha A_{\min}}{\nu\frac{Q}{\delta} + \mu} = C_{\min}.$$

Lastly, from Eq. (5) we have

$$\frac{\mathrm{d}E}{\mathrm{d}t} \geqslant Q - \left(\delta + \tau\frac{\Lambda}{\mu}\right)E,$$

this implies that

$$\lim_{t\to\infty} \inf E(t) \geqslant \frac{Q}{\delta + \tau\frac{\Lambda}{\mu}} = E_{\min}.$$

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Considering that $0 \leqslant N \leqslant \Lambda/\mu$ and $0 \leqslant E \leqslant Q/\delta$, we have

$$T_{\min} \leqslant \lim_{t\to\infty} \inf T(t) \leqslant \lim_{t\to\infty} \sup T(t) \leqslant \frac{\Lambda}{\mu},$$
$$S_{\min} \leqslant \lim_{t\to\infty} \inf S(t) \leqslant \lim_{t\to\infty} \sup S(t) \leqslant \frac{\Lambda}{\mu},$$
$$C_{\min} \leqslant \lim_{t\to\infty} \inf C(t) \leqslant \lim_{t\to\infty} \sup C(t) \leqslant \frac{\Lambda}{\mu},$$
$$A_{\min} \leqslant \lim_{t\to\infty} \inf A(t) \leqslant \lim_{t\to\infty} \sup A(t) \leqslant \frac{\Lambda}{\mu},$$
$$E_{\min} \leqslant \lim_{t\to\infty} \inf E(t) \leqslant \lim_{t\to\infty} \sup E(t) \leqslant \frac{Q}{\delta}.$$

Since each component T, S, A, C, E uniformly persists given that the proportion of human recruitment with family history does not exceed one (not all human recruits have family background), then system (1)–(5) is uniformly persistent, implying the survival of all population in future if they initially existed in the system. □

4 Simulation of the model

In this section, numerical simulation of system (1)–(5) is carried out to explain the applicability of the analytical results and the behaviour of system (1)–(5) under different scenarios, using the ode45 solver based on the Runge–Kutta method in MATLAB software packages under the set of parameters in Table 1.

Using the set of parameter values given in Table 1, simulation of system (1)–(5) showed that the unique positive equilibrium point x exists and is given by $\widetilde{T} = 6, \widetilde{S} = 35890, \widetilde{A} = 50820, \widetilde{C} = 117450, \widetilde{E} = 240$. In addition, conditions of local stability (11), global stability (12) and uniform persistence are satisfied.

Table 1. Description and value of parameters used in the model system (1)–(5). The parameters are given per day.

Parameter	Definition	Value	Reference
ρ	Proportion of human recruitment with family history	0.00031	[12]
Λ	Per capita recruitment rate of human population	10	Assumed
β	Effective exposure rate of susceptible to polluted environment	0.000002	[8]
μ	Natural mortality rate of human population in Uganda	0.000044	[22]
γ	Modification parameter for asthma development in low genetic risk susceptible	0.48	Assumed
ν	Recurring rate of asthma	0.0000005	Assumed
σ	Asthma related death rate in Uganda	0.00002	[23]
α	Average rate of asthma control	0.00096	[15]
δ	Natural depletion rate of environmental pollutant	0.001	[8]
τ	Human dependent depletion rate of pollutant	0.000002	[8]
ω	Probability of success of asthma control through public health education/awareness	0.4	Assumed
Q	Rate of emission of pollutant into the environment in Uganda	100 $\mu g/m^3$	[11]

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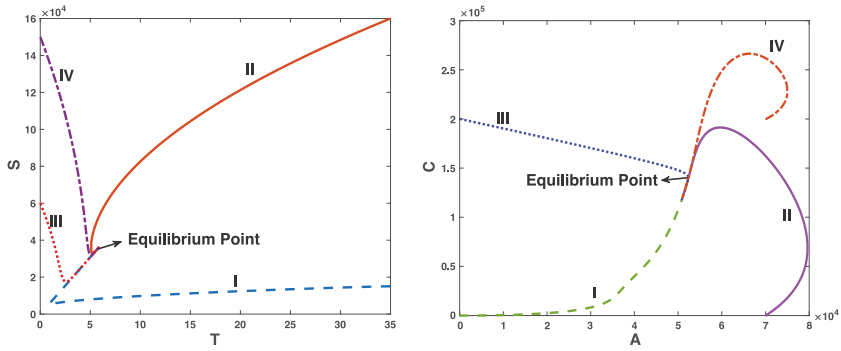
4.1 Global stability of unique positive equilibrium point

Figures 1(a) and 1(b) have been plotted to show the global stability behaviour of unique positive equilibrium point x. It is noted that all solution trajectories initiating from interior of the region of attraction tend towards the equilibrium point. This means that the unique positive equilibrium point x is globally asymptotically stable in agreement with Theorem 2.

4.2 Effect of pollutant reduction strategies

In the model, the effect of pollutant reduction is through decreasing the pollutant emitting rate parameter, Q, into the environment. In other words, a reduction in Q leads to reduction in the concentration of pollutant in the environment. Thus, Q can be used to determine the effect of pollutant reduction on the development and recurrence of asthma. As a result, Fig. 2 shows the time course of all the densities; susceptible with family history (high genetic risk), T, susceptible without family history (low genetic risk), S, asthmatic, A, controlled asthmatic, C, and concentration of environmental pollutant, E, when Q is varied. It is clear from Fig. 2 that low cumulative rate of emission of pollutant into the environment corresponds to a reduction in disease prevalence. Also, note that the density of T is much more affected by Q as compared to the density of S.

Similarly, Fig. 3 depicts results as varying values of β and ν on the equilibrium levels of $\widetilde{T}$, $\widetilde{S}$, $\widetilde{A}$, $\widetilde{C}$ and $\widetilde{E}$ are carried out. When β and ν are both zero, the equilibrium levels of $\widetilde{T}$, $\widetilde{S}$ are at their maximum, while that of $\widetilde{A}$, $\widetilde{C}$ and $\widetilde{E}$ are at their minimum. As the values of β and ν increase simultaneously, the equilibrium levels of susceptible ($\widetilde{T}$, $\widetilde{S}$) decrease whereas that of asthmatics ($\widetilde{A}$, $\widetilde{C}$) and environmental pollutant ($\widetilde{E}$) increase. This means that more effective exposure of susceptible to polluted environment (corresponding to



(a) (b)

Figure 1. Simulation of system (1)–(5), global stability of unique positive equilibrium point x in susceptible with low genetic risk (S), susceptible with high genetic risk (T)-space (a) and in controlled asthmatic (C), asthmatic (A)-space (b). All parameters are fixed on their baseline values in Table 1 for different initial conditions I: $[35, 15000, 50, 19, 100]$, II: $[35, 160000, 0, 0, 2000]$, III: $[0, 60000, 0, 90, 600]$, IV: $[0, 150000, 500, 10, 100]$ (a) and I: $[200, 50000, 0, 0, 800]$, II: $[200, 400000, 70000, 0, 200]$, III: $[0, 50000, 0, 200000, 1000]$, IV: $[0, 400000, 70000, 200000, 400]$ (b).

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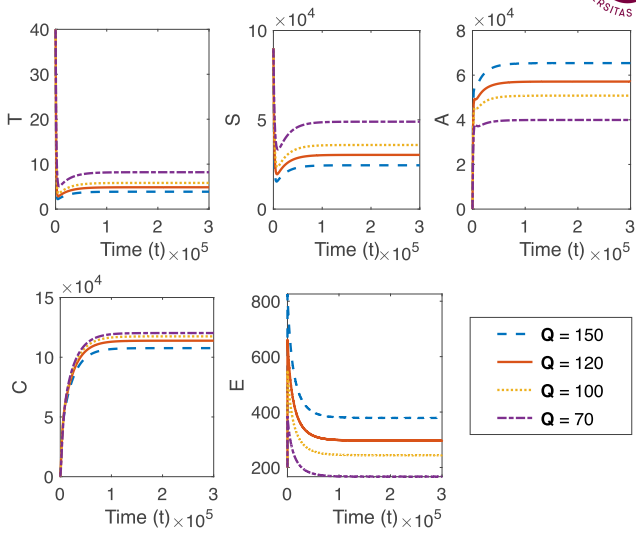

Figure 2. Simulations of system (1)–(5), showing a time course of susceptible with high genetic risk (T), susceptible with low genetic risk (S), asthmatic (A), controlled asthmatic (C) and concentration of environmental pollutant (E) as emission rate of environmental pollutant (Q) is varied and other parameters are fixed on their baseline values in Table 1. Assumed initial conditions: $T = 40$, $S = 90000$, $A = 0$, $C = 0$, $E = 200$.

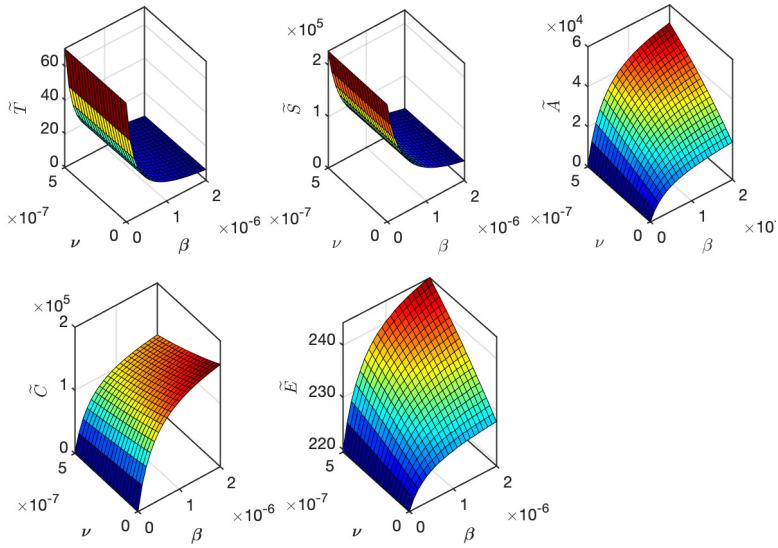

Figure 3. The equilibrium level of susceptible with high genetic risk ($\widetilde{T}$), susceptible with low genetic risk ($\widetilde{S}$), asthmatic ($\widetilde{A}$), controlled asthmatic ($\widetilde{C}$) and concentration of environmental pollutant ($\widetilde{E}$) respectively as a function of different values of β (corresponding to effective exposure rate of susceptible to polluted environment) and ν (representing the recurring rate of asthma due to polluted environment) as remaining parameters are same as given in Table 1.

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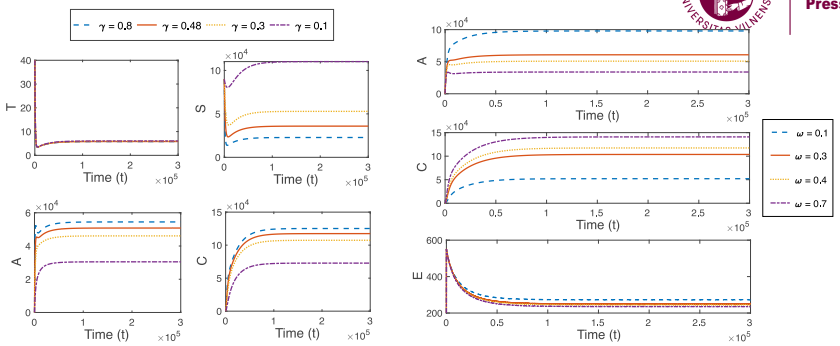

(a) (b)

Figure 4. (a) Simulations of system (1)–(5), showing a time course of susceptible with high genetic risk (T), susceptible with low genetic risk (S), asthmatic (A), controlled asthmatic (C) as modification parameter for asthma development in low genetic risk susceptible (γ) is varied. Assumed initial conditions: $T = 40$, $S = 90000$, $A = 0$, $C = 0$. (b) Simulations of system (1)–(5), showing a time course of asthmatic (A), controlled asthmatic (C) and concentration of environmental pollutant (E) as a function of probability of success of asthma control through public health education/awareness (ω). Assumed initial conditions: $A = 0$, $C = 0$, $E = 200$. Other parameters are fixed on their baseline values in Table 1.

higher values of β) and more asthma recurrence (corresponding to higher values of ν) result into rise in asthmatic population thus persistence of asthma in the population. In other words, a sufficiently effective pollutant reduction strategy resulting into low effective exposure to pollutant and asthma recurrence can lead to reduction of the asthmatic and increase in the controlled asthmatic in the population.

In addition, as asthma development in low genetic risk class increases (corresponding to $\gamma \to 1$) due to more exposure to highly polluted environment (with the assumption that asthma development rate in low and high genetic classes is the same), disease prevalence increases, thus, the need to avoid polluted environment is seen in Fig. 4(a).

4.3 Effect of public health education/awareness control strategies

The goal here is to assess the effect of public health education/awareness on control of asthma by reducing and avoiding polluted environment as a way of enhancing asthma control. In the model, public health education/awareness control strategy is incorporated by rescaling the asthma control coefficient from α to $\omega\alpha$. Simulations were carried out with varying values of ω (Fig. 4(b)); obtained results show an increase in the density of asthmatic and reduction in the controlled asthmatic with increasing non-compliance to public health education/awareness (smaller values of ω). However, increase in public health education/awareness and compliance results into more controlled asthmatic population.

4.4 Effect of public health education/awareness with human dependent pollutant depletion strategies

Figure 5 shows the cumulative effect of ω and τ on the equilibrium levels of $\widetilde{T}$, $\widetilde{S}$, $\widetilde{A}$, $\widetilde{C}$ and $\widetilde{E}$. It is attributed that as values of ω and τ increase concurrently, the equilibrium

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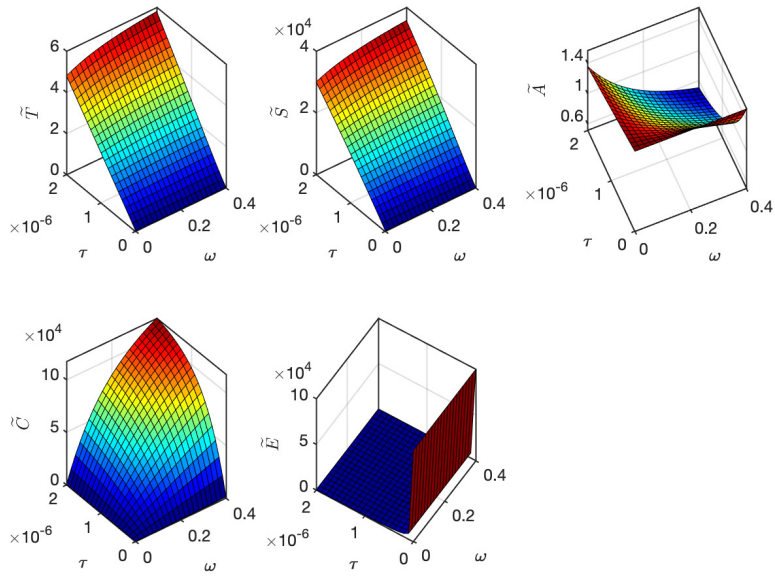

Figure 5. Variation of equilibrium values of susceptible with high genetic risk ($\widetilde{T}$), susceptible with low genetic risk ($\widetilde{S}$), asthmatic ($\widetilde{A}$), controlled asthmatic ($\widetilde{C}$) and concentration of environmental pollutant ($\widetilde{E}$) for different values of ω (probability of success of asthma control through public health education/awareness) and τ (human dependent depletion rate of pollutant) as other values of parameters are same as given in Table 1.

levels of the susceptible ($\widetilde{T}$ and $\widetilde{S}$) and controlled asthmatic ($\widetilde{C}$) increase, while that of asthmatic ($\widetilde{A}$) and environmental pollutant ($\widetilde{E}$) decrease. Effective public health education/awareness (higher values of ω) together with continuous depletion of environment pollutant by human population (higher values of τ) show a marked increase and reduction in the controlled asthmatic and asthmatic population, respectively, as well as a reduction in environmental pollutant. Therefore, a combination of more compliance to public health education/awareness together with implementation of human dependent depletion of environmental pollutant can significantly control asthma and its development in the population.

5 Discussion and future study

The development of asthma model was formulated and analysed in a polluted environment and among human population with and without genetic risk factor. The model incorporates essential factors towards asthma development, recurrence of asthma and examines various asthma control strategies i.e. pollutant reduction strategy, public health education/awareness to control development and recurrence of asthma. Conditions for the existence and stability behaviour using Lyapunov functional approach of the unique coexistence equilibrium point are obtained, and permanence influence of the system is assessed. The model was used to assess the impact of environmental pollutant emission rate, Q, effective exposure rate to environmental pollutant of susceptible and controlled

class, β, asthma recurrence rate, ν, pollutant reduction strategies, public health education/awareness and human dependent pollutant depletion towards asthma development and control dynamics.

The analysis of the model illustrates that rise in rate of pollutant emission into the environment fuels the effective exposure rate of susceptible to polluted environment and asthma recurrence, markedly keeping asthma prevalence at higher levels. In addition, numerical simulations qualitatively indicate that a combination of more effective exposure of susceptible to polluted environment (higher values of β) and more asthma recurrence (higher values of ν) result into extreme rise in asthma development and its persistence in the population. More so, disease prevalence increases with more exposure of susceptible with low genetic risk to highly polluted environment ($\gamma \to 1$). Therefore, effective preventive strategies (pollutant reduction and control strategies) that lower Q, β, ν or γ, lead to reduction in the concentration of environmental pollutant and, consequently, lessen the development and recurrence of asthma in the population.

The obtained results show that this (reduction in the concentration of environmental pollutant that lessens asthma development and recurrence in the population) can easily be achieved when public health education/awareness, human dependent depletion of environmental pollutant are implemented. Additionally, intensifying compliance to public health education/awareness of asthma together with continuous human dependent depletion of environmental pollutant play a significant role in keeping asthma development and recurrence to its barest minimal, thus, increasing controlled asthmatic in the population.

The mathematical model can be extended further by considering logistic function or holing type functional response of human population exposure to concentration of environmental pollutant that yields to a chance of asthma development. Also, there are seasons in which pollutant is more intense in the environment; thus, the effect of seasonality can be incorporated in the model.

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