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Full Length Research Article

ANALYSIS OF MATHEMATICAL MODEL FOR ASTHMA CAUSED BY THE EFFECTS OF ENVIRONMENTAL POLLUTION

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ABSTRACT
Asthma is a condition in which your airways narrow and swell and may produce extra mucus. In this study, an improved mathematical model of asthma which includes infected class not detected, infected detected class and recovered (relieved) class was formulated. The model exhibits two equilibrium, namely disease free equilibrium and endemic equilibrium. The analysis of positivity and boundedness of solutions showed that the model is epidemiologically well pose d. Simulation results showed that when pollutants are discharged into the environment at a constant rate, the asthma disease cases also increase in the population due to the fact that the interaction rate of both susceptible and infected undetected with pollutants in creases.Finally, asthma can be control by restricting the smokers from the population and the rate of release of pollutants into the environment.

Keywords: Asthma, model formulation disease free equilibrium, endemic equilibrium

IINTRODUCTION
Asthma is an inflammatory disease affecting the airways that carry air to and from the lungs. People who suffer from this chronic condition are said to be asthmatic. Some mathematical modeling on asthma has been done such as in (Ghosh, 2011; Amory et al., 2012; Wild et al., 2005; Shakirova et al., 2013; Stanciole et al., 2012; Omoloye & Adewale, 2021) Analyzed some mathematical models to investigate the spread of asthma due to inhaled pollutants from industry. to considered two types of demographics namely population with constant immigration and population with logistic growth and considered three cases, regarding the release of pollutant into the environment. (Burrowes *et al.*, 2013; Chatzimichail *et al*., 2013; Hanifi, 2013; Alejandra *et al*., 2020) Predicted a new method for asthma outcome, based on principal component analysis and Least Square Support Vector Machine Classifier which consists of three stages namely principal component analysis, the pattern classification and classification accuracy and 10-fold cross-validation, their proposed prediction system can be used in asthma outcome prediction and that some risk factors enhance its predictive ability. (Yangyin et al., 2013; Alkhudhari et al, 2014) proposed a mathematical model and analyzed the behavior of smoking dynamics in a population with peer pressure effect on temporary quitters and the research result shows that the smokers' population reaches a steady state of approximately 6% of the total population. (David, 2015; Ana et al., 2016; Charles et al., 2016; Winkler et al., 2016; Kavitha, 2016) explained that biometeorology, emerged as an interdisciplinary field dedicated to the study of the mutual influence between living things and the surrounding environment. They also explained that the Asthma is linked to an out of control reaction from the immune system triggered by a diversity of pathways. (Junehyuk et al., 2017; Joseph et al., 2018; Hutapea et al., 2018; Evgeniy et al., 2018; Tri Andri et al., 2018; Qinghua & Xiantao, 2019; Omoloye et al., 2020; Emmanuel et al., 2023). Having gone through all these literature review not much work has been done especially in this area (Asthma), therefore the work of Naresh and Tripathi (2009) was extended to come up with a new mathematical model of Asthma by incorporating the infected class not detected, infected detected class and the recovered (relieved) class in their model.
Nareth and Tripathi Model (2009)

$$\left.\begin{aligned}
\frac{dS}{dt} &= \wedge - \mu S - \gamma SP - \beta SC \\
\frac{dE}{dt} &= \gamma SP + \beta SC - \lambda_1 EP - \lambda_2 EC - \mu E \\
\frac{dI}{dt} &= \lambda_1 EP + \lambda_2 EC + \theta_1 C + \theta_2 CP - \alpha I - \mu I \\
\frac{dC}{dt} &= Q - \theta_1 C - \theta_2 CP - \theta C - \mu C \\
\frac{dP}{dt} &= A(N) - \tau P + qC
\end{aligned}\right\} \quad (1)$$

Descriptions of New Model
A modify mathematical model that consider the causes of asthma due to effect of environmental pollutants was developed. The total population is divided into seven classes; susceptible individuals $S(t)$, smokers class $C(t)$, exposed class $E(t)$, infected undetected $I_U(t)$, infected detected $I_D(t)$, recovered (relieved) class $R(t)$ and one environmental pollution class $P(t)$ .Therefore the total population of human is denoted by;

$$N(t) = S(t) + C(t) + E(t) + I_U(t) + I_D(t) + R(t) \quad (2)$$

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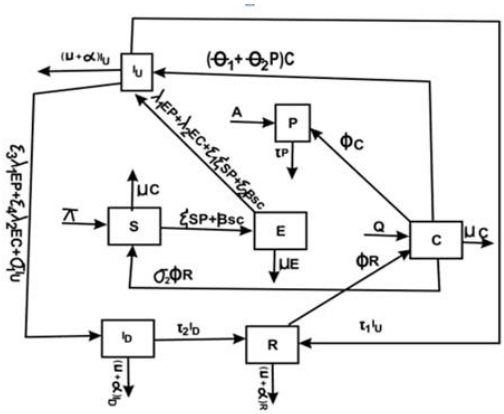

Figure.1. Schematic diagram of the model

The rate of change of susceptible population is given by;

$$\frac{dS}{dt} = \pi - \mu S - \xi SP - \beta SC + \sigma_3 \phi R \qquad (3)$$

The recruitment rate of susceptible individuals to the population (π), while the natural death rate affect the susceptible population (μS), the transmission coefficient of the susceptible to the environment pollution affect the susceptible population (ξSP) and another transmission coefficient of the susceptible to the smokers due to their relationship also affected the susceptible population (βSC), fraction of recovered were moved back to the susceptible class and the rate at which moves back to susceptible and smokers class.

Hence,

$$\frac{dC}{dt} = Q - \theta_1 C - \theta_2 CP - \theta C - \mu C + (1-\sigma_2)\phi R \qquad (4)$$

The population of smokers class is increases by smokers recruitment rate (Q), but the population decreases by the rate at which smokers become infected ($\theta_1 C$)and the rate at which smokers came into contact with the disease ($\theta_2 CP$), it also decreases due to those that quit smoking (θC) decreases by natural death rate of the smokers (μC) and increases by the rate of those that recovered back to the smokers class (ϕR), decreases by the fraction of recovered that moves back to susceptible ($\sigma_2 \phi R$).

$$\frac{dE}{dt} = (1-\varepsilon_1)\xi SP + (1-\varepsilon_2)\beta SC - \lambda_1 EP - \lambda_2 EC - \mu E \qquad (5)$$

Also, the transmission coefficients of the exposed class, due to the interaction of susceptible class, with the environmental pollution is increased (ξSP) and decreases by the fraction of the transmission rate of susceptible to infected undetected, due to interaction with the pollutants ($\varepsilon_1 \xi SP$) , it also increases due to the transmission coefficient from susceptible to exposed, due to interaction with smokers (βSC) and decreases due to the fraction of the transmission rate susceptible to exposed, due to interaction with smokers ($\varepsilon_1 \beta SC$), decreases by the transmission rate of exposed, due to the interaction of exposed with pollutants ($\lambda_1 EP$), decreases by the transmission rate of exposed, due to the interaction of exposed with smokers, and finally decreases by the naturally death rate (μE).

$$\frac{dI_U}{dt} = \varepsilon_1 \xi SP + \varepsilon_2 \beta SC + (\theta_1 + \theta_2 P)C + (1-\varepsilon_4)\lambda_2 EC - (\sigma_2 + \tau_1 + \mu + \alpha)I_U \qquad (6)$$

The infected undetected population is increased by the transmission coefficients from susceptible due to the interaction with pollutants and a fraction of the transmission rate of the susceptible to infected undetected due to interaction with pollutant ($\varepsilon_1 \xi SP$), it also increases by both the transmission coefficients from susceptible due to the interaction with smokers and fraction of transmission rate of susceptible to infected undetected due to interaction with smokers ($\varepsilon_2 \beta SC$), also increases by the rate at which smokers become infected ($\theta_1 C$) and the rate at which smokes came into contact due to the pollutants ($\theta_2 PC$), increases the transmission rate of the exposed to infected undetected ($\lambda_2 EC$) and decreases by the transmission rate of exposed due to the interaction with the smokers $(1-\varepsilon_4)\lambda_2 EC$, also decreases by fraction of recovered that moves back to susceptible, to the recovered class, due to natural death and disease induced death rate ($\sigma_2 + \tau_1 + \mu + \alpha)I_U$.

$$\frac{dI_D}{dt} = \varepsilon_3 \lambda_1 EP + \varepsilon_4 \lambda_2 EC + \sigma_2 I_U - (\tau_2 + \mu + \alpha)I_D \qquad (7)$$

The infected detected population is increased by the transmission rate of exposed due to the interaction with the pollutants $(\varepsilon_3 \lambda_1 EP)$, also increases by the transmission rate exposed due to the interaction with smokers $(\varepsilon_4 \lambda_2 EC)$, and increases by the fraction of recovered from infected undetected $(\sigma_2 I_U)$, it decreases by the rate at which infected detected moves to recovered and both natural death rate and disease induced death rate.

$$\frac{dR}{dt} = \tau_1 I_U + \tau_2 I_D - (\phi + \mu + \alpha)R \qquad (8)$$

The recovered (relieved) population increases by the rate at which infected undetected and infected detected moves to recovered class $\tau_1 I_U + \tau_2 I_D$, it decreases by the rate at which recovered moves back to susceptible also decreases by natural death rate and disease induced death rate.

$$\frac{dP}{dt} = A - \tau P + qC \qquad (9)$$

This is not human population but cumulative concentration of

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pollutants where (A) is the cumulative density of pollutants, the pollutants is decreases by the depletion rate of the environmental discharge when environment is clean (τP), the pollutants increases again due to the rate at which pollution increase due to smokers smoke (qC).

Putting all the above formulations and assumptions together the following system of differential equations were obtained.

$$\left.\begin{aligned}
\frac{dS}{dt} &= \pi - \mu S - \xi SP - \beta SC + \sigma_2 \phi R,\\
\frac{dC}{dt} &= Q - \theta_1 C - \theta_2 CP - \theta C - \mu C + (1-\sigma_2)\phi R,\\
\frac{dE}{dt} &= (1-\varepsilon_1)\xi SP + (1-\varepsilon_2)\beta SC - \lambda_1 EP - \lambda_2 EC - \mu E,\\
\frac{dI_U}{dt} &= \varepsilon_1 \xi SP + \varepsilon_2 \beta SC + (\theta_1 + \theta_2 P)C + (1-\varepsilon_3)\lambda_1 EP + (1-\varepsilon_4)\lambda_2 EC - (\sigma_1 + \tau_1 + \mu + \alpha) I_U,\\
\frac{dI_D}{dt} &= \varepsilon_3 \lambda_1 EP + \varepsilon_4 \lambda_2 EC + \sigma_1 I_U - (\tau_2 + \mu + \alpha) I_D,\\
\frac{dR}{dt} &= \tau_1 I_U + \tau_2 I_D - (\phi + \mu + \alpha) R,\\
\frac{dP}{dt} &= A - \tau P + qC.
\end{aligned}\right\} \quad (10)$$

Together with initial conditions $S(0)>0, C(0)>0, E(0)>0, I_U(0)>0, I_D(0)>0, R(0)>0$

Table1. Description of variables of Asthma models

Variables	Description
N	Total population
S	Susceptible class
C	Smokers class
E	Exposed class
I_U	Infected class but not detected
I_D	Infected and detected class
R	Recovered class
P	Cumulative concentration of pollutants

Table 2. Description of parameters of Asthma models

Parameters	Definitions
π	Recruitment rate
μ	Natural death rate
A	Cumulative density of pollutants
ξ	Transmission coefficients from susceptible to exposed and infected undetected due to interaction with pollutants.
β	Transmission coefficient from susceptible to exposed and infected undetected due to interaction with smokers
σ_3	Fraction of recovered that moves back to susceptible
θ	Rate at which smokers quit smoking
q	Rate at which pollution increase due to smokers smoking
τ	Depletion rate of environmental discharge as environment cleans
λ_1	Transmission rate of exposed to infected u ndetected and infected undetected due to interaction with pollutants
λ_2	Transmission rate of exposed to infected u ndetected and infected undetected due to interaction with smokers
θ_1	Rate at which smokers become infected
θ_2	Rate at which smokers came into contact
$\varepsilon_1, \varepsilon_2, \varepsilon_3, \varepsilon_4$	Fraction of transmission rate from exposed
ϕ	Progression rate from recovered to susceptible and smokers class
τ_1	Rate at which infected undetected moves to recovered class
τ_2	Rate at which infected detected moves to recovered class
Q	Smokers recruitment rate
α	Disease induced death rate

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Positivity and Boundedness of Solutions of Asthma Model

For the Asthma model (10) to be epidemiological meaningful, it is important to prove that all solutions with non-negative initial data will remain non-negative for all time $t \geq 0$

Theorem 1

If S(0), C(0), E(0), $I_U(0), I_D(0), R(0), P(0)$ are non-negative, the solutions S, C, E, I_U, I_D, R, P of the Asthma disease model (10) are non-negative for all $t \geq 0$

Proof.

Consider the biological-feasible region. $\prod = \left\{ (S, C, E, I_U, I_D, R, P) \varepsilon \Re_+^7 : N \leq \frac{\pi}{\mu} \right\}$, it will be proved that $\prod$ is positively invariant.

The total population of Asthma disease transmission model is obtained by adding the model equation (10) and is given by;

$$\frac{dN}{dt} = \pi + Q - \theta C - \mu N - (I_U + I_D)\alpha \qquad (11)$$

Setting the disease induced death to zero in equation (11), then equation (11) becomes

$$\frac{dN}{dt} \leq \pi - \mu N \qquad (12)$$

By separation of variable method, equation (12) become,

$$\frac{dN}{\pi - \mu N} \leq dt \qquad (13)$$

Integrating both sides of equation (13) to obtain equation (14),

$$\int \frac{dN}{\pi - \mu N} \leq \int dt \qquad (14)$$

$\frac{-1}{\mu} In(\pi - \mu N) \leq t + C$, which can be re-write as in equation (15) given below

$\pi - \mu N \geq Ae^{-\mu t}$, where A is constant of integration (15)

Applying the initial condition $N(0) = N_0$ in equation (15), to obtain

$A = \pi - \mu N$ which upon plugging into equation (15), yields

$$\pi - \mu N \geq (\pi - \mu N_0)e^{-\mu} \qquad (16)$$

Then by rearranging equation (16) to obtain equation (17)

$$N \leq \frac{\pi}{\mu} - \left[\frac{\pi - \mu N_0}{\mu} \right] e^{-\mu t} \qquad (17)$$

As $t \to \infty$ in equation (17) the population size $N \to \frac{\pi}{\mu}$ which implies that

$0 \leq N \leq \frac{\pi}{\mu}$. Thus the feasible solution set of system equation of the model enter and remain in the region.

$$\prod = \left\{ (S, C, E, I_U, I_D, R) \varepsilon \Re_+^6 : N \leq \frac{\pi}{\mu} \right\}$$

Therefore, the basic model is epidemiologically well posed. Hence it is sufficient to study the dynamics of the basic model in $\prod = \left\{ (S, C, E, I_U, I_D, R) \varepsilon \Re_+^6 : N \leq \frac{\pi}{\mu} \right\}$.

Existence of Disease Free Equilibrium (DFE) of Asthma Disease Model

Disease free equilibrium point is the steady state solution where there is no infection in the population that is; (C = E = I_U = I_D = R= 0) .After some manipulations we obtain

$$E_0 = (S, C, E, I_U, I_D, R, P) = \left(\frac{\pi}{\mu}, 0,0,0,0,0,0 \right)$$

Existence of Endemic Equilibrium Point (EEP) of Asthma Disease Model

$$S \neq C \neq E \neq I_U \neq I_D \neq R \neq P \neq 0$$

$$\left.\begin{aligned}
S' &= \pi - \mu S - \xi SP - \beta SC + \sigma_2 \phi R = 0, \\
C' &= Q - \theta_1 C - \theta_2 CP - \theta C - \mu C + (1 - \sigma_2)\phi R = 0, \\
E' &= (1 - \varepsilon_1)\xi SP + (1 - \varepsilon_2)\beta SC - \lambda_1 EP - \lambda_2 EC - \mu E = 0, \\
I_U' &= \varepsilon_1 \xi SP + \varepsilon_2 \beta SC + (\theta_1 + \theta_2 P)C + (1 - \varepsilon_3)\lambda_1 EP + (1 - \varepsilon_4)\lambda_2 EC - (\sigma_1 + \tau_1 + \mu + \alpha)I_U = 0, \\
I_D' &= \varepsilon_3 \lambda_1 EP + \varepsilon_4 \lambda_2 EC + \sigma_1 I_U - (\tau_2 + \mu + \alpha)I_D = 0, \\
C' &= Q - \theta_1 C - \theta_2 CP - \theta C - \mu C + (1 - \sigma_2)\phi R = 0, \\
R' &= \tau_1 I_U + \tau_2 I_D - (\phi + \mu)R = 0, \\
P' &= A - \tau P + qC = 0.
\end{aligned}\right\} \qquad (18)$$

From (18) $A - \tau P + qC = 0$ after simplification we have

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$$P = \frac{A + qC}{\tau} \quad (19)$$

Let $\varepsilon_1 = \varepsilon_2 = \varepsilon_3 = \varepsilon_4 = \sigma_3 = 1$

We progress from one compartment to another compartment together without any fraction being left behind. And q = θ = 0 Therefore $P = \frac{A}{\tau}$ (20)

Substituting the conditions, it becomes

$$S = \pi - \mu S - \xi SP - \beta SC \quad (21)$$

$$C = Q - \theta_1 C - \theta_2 CP - \theta C - \mu C + \phi R \quad (22)$$

$$E = \xi SP + \beta SC - \lambda_1 EP - \lambda_2 EC - \mu E \quad (23)$$

$$I_U = \theta_1 C + \theta_2 CP + \lambda_1 EP + \lambda_2 EC - d_1 I_U \quad (24)$$

$$I_D = \sigma_1 I_U - d_2 I_D \quad (25)$$

$$R = \tau_1 I_U + \tau_2 I_D - d_3 R \quad (26)$$

$$P = A - \tau P + qC \quad (27)$$

Let $d_1 = (\sigma_1 + \tau_1 + \mu + \alpha)$, $d_2 = (\tau_2 + \mu + \alpha)$, $d_3 = \phi + \mu$, $d_4 = \theta_1 + \mu$

From (24) $\sigma_1 I_U - d_2 I_D = 0$

, after simplification we have $I_D^* = \frac{\sigma_1}{d_2} I_U$ (28)

From (26) $\tau_1 I_U + \tau_2 I_D - d_3 R = 0$, after manipulation we have $R^* = \frac{(\tau_1 d_2 + \tau_2 \sigma_1) I_U}{d_2 d_3}$ (29)

From (22), $Q - \theta_1 C - \theta_2 CP - \theta C - \mu C + \phi R = 0$ upon the simplification, this was obtained

$$C^* = \frac{\tau(Qd_2 d_3 + \phi(\tau_1 d_2 + \tau_2 \sigma_1) I_U)}{d_2 d_3 (d_4 \tau + \theta_2 A)} \quad (30)$$

From (21), $\pi - \mu S - \xi SP - \beta SC$ = 0. After some simplification we obtain;

$$S^* = \frac{\pi\tau(d_2 d_3 + \phi(\tau_1 d_2 + \tau_2 \sigma_1)) I_U}{\mu + \xi A + \beta\tau(Qd_2 d_3 + \phi(\tau_1 d_2 + \tau_2 \sigma_1)) I_U} \quad (31)$$

From (23) $\xi SP + \beta SC - \lambda_1 EP - \lambda_2 EC - \mu E = 0 \quad (\xi P + \beta C) S = (\lambda_1 P + \lambda_2 C + \mu) E$

$$\frac{(\xi P + \beta C)\pi\tau + (d_2 d_3 + \phi(\tau_1 d_2 + \tau_2 \sigma_1))}{\mu + \xi A + \beta\tau(Qd_2 d_3 + \phi(\tau_1 d_2 + \tau_2 \sigma_1) I_U} = (\frac{\lambda_1 A}{\tau} + \lambda_2 C + \mu) E$$

$$\frac{(\xi P + \beta C)\pi\tau(d_2 d_3 + \phi(\tau_1 d_2 + \tau_2 \sigma_1))}{\mu + \xi A + \beta\tau(Qd_2 d_3 + \phi(\tau_1 d_2 + \tau_2 \sigma_1) I_U)} = \left(\frac{\lambda_1 A}{\tau} + \frac{\lambda_2 \tau(Qd_2 d_3 + \phi(\tau_1 d_2 + \tau_2 \sigma_1) I_U)}{d_2 d_3 (d_4 \tau + \theta_2 A)} + \mu \right) E$$

E^* =

$$\frac{\xi A + \beta\tau(Qd_2 d_3 + \phi(\tau_1 d_2 + \tau_2 \sigma_1) I_U)\pi\tau(d_2 d_3 + \phi(\tau_1 d_2 + \tau_2 \sigma_1))}{\tau + d_2 d_3 (d_4 \tau + \theta_2 A)(\mu + \xi A + \beta\tau(Qd_2 d_3 + \phi(\tau_1 d_2 + \tau_2 \sigma_1) I_U} \times \frac{\tau(d_2 d_3 (d_4 \tau + \theta_2 A))}{\lambda_1 A(d_2 d_3 (d_4 \tau + \theta_2 A) + \lambda_2 \tau^2 (Qd_2 d_3 + \phi)(\tau_1 d_2 + \tau_2 \sigma_1) I_U + \mu\mu\tau}$$

Let $d_2 d_3 (d_4 \tau + \theta_2 A) = X$ and $Qd_2 d_3 + \phi(\tau_1 d_2 + \tau_2 \sigma_1) = Y$

$$E^* = \frac{\xi A + \beta\tau Y I_U \pi\tau X}{\tau + X(\mu + \xi A + \beta\tau Y I_U)} \times \frac{\tau}{\lambda_1 A + \lambda_2 \tau^2 Y I_U + \mu X}$$

$$E^* = \frac{\tau(\xi A + \beta\tau^2 Y \pi X I_U)}{(\tau + X(\mu + \xi A + \beta\tau Y I_U))(\lambda_1 A + \lambda_2 \tau^2 Y I_U + \mu X)} \quad (32)$$

From (24) $\theta_1 C + \theta_2 CP + \lambda_1 EP + \lambda_2 EC - (\sigma_1 + \tau_1 + \mu + \alpha) I_U$ = 0

$$\theta_1 C + \theta_2 CP + \lambda_1 EP + \lambda_2 EC - d_1 I_U = 0$$

$$(\theta_1 + \theta_2 P) C + (\lambda_1 P + \lambda_2 C) E = d_1 I_U \quad (33)$$

Substituting P into equation (33), it becomes

$$(\theta_1 + \frac{\theta_2 A}{\tau}) C + (\frac{\lambda_1 A}{\tau} + \frac{\lambda_2 \tau(Qd_2 d_3 + Q(\tau_1 d_2 + \tau_2 \sigma_1) I_U}{d_2 d_3 (d_4 \tau + \theta_2 A)}) E = d_1 I_U$$

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$$(\frac{\theta_1\tau+\theta_2 A}{\tau})C+(\frac{\lambda_1 A(d_2d_3(d_4\tau+\theta_2 A))+\lambda_2\tau^2(Qd_2d_3+\phi(\tau_1d_2+\tau_2\sigma_1)I_U}{\tau(d_2d_3(d_4\tau+\theta_2 A))})E=d_1I_U$$

Simplifying:

$$I_U^*=\frac{(\theta_1\tau+\theta_2 A)\tau(Qd_2d_3+\phi(\tau_1d_2+\tau_2\sigma_1)I_U+(\lambda_1 A(d_2d_3(d_4\tau+\theta_2 A))+\lambda_2\tau^2(Qd_2d_3+\phi(\tau_1d_2+\tau_2\sigma_1)I_U)\tau(\xi A+\beta\tau^2YX\pi I_U)}{d_1\tau(d_2d_3(d_4\tau+\theta_2 A)(\tau+X(\mu+\xi A+\beta\tau YI_U)(\lambda_1 A+\lambda_2\tau_2 YI_U+\mu X))}$$

Through cross multiplication

$$I_U^*(d_1\tau(d_2d_3(d_4\tau+\theta_2 A))(\tau+X(\mu+\xi A+\beta\tau YI_U)(\lambda_1 A+\lambda_2\tau_2 YI_U+\mu X))=(\theta_1\tau+\theta_2 A)\tau(Qd_2d_3+\phi(\tau_1d_2+\tau_2\sigma_1)I_U$$
$$+(\lambda_1 A(d_2d_3(d_4\tau+\theta_2 A)))+\lambda_2\tau^2(Qd_2d_3+\phi(\tau_1d_2+\tau_2\sigma_1)I_U)\tau(\xi A+\beta\tau^2YX\pi I_U)$$

Expanding all the brackets and re-arranging it, it becomes

$$(d_1d_2d_3d_4\tau^3\beta XY^2\lambda_2\tau_2+d_1d_2d_3\tau^2\theta_2 A\beta XY^2\lambda_2\tau_2)I_U^3+(d_1d_2d_3d_4\tau^2\xi\lambda_2\tau_2Y+d_1d_2d_3d_4\tau^4\mu X\lambda_2Y+d_1d_2d_3d_4\tau^3\beta X$$
$$Y\lambda_1A+d_1d_2d_3d_4\tau^3\beta X^2Y\mu+d_1d_2d_3\tau\theta_2A\xi\lambda_2\tau_2Y+d_1d_2d_3\tau\theta_2A\mu X\lambda_2\tau_2Y+d_1d_2d_3\tau^2\theta_2A^2\beta XY\lambda_1+d_1d_2d_3\tau^2\theta_2A\beta$$
$$X^2Y\mu)I_U^2+(d_1d_2d_3(d_4\tau^2\xi\lambda_1A+d_4\tau^2\xi\mu X+d_4\tau^2\mu X\lambda_1A+d_4\tau^2\mu X^2+d_4\tau^2\xi A^2X\lambda_1+d_4\tau^2\xi A\mu X^2+d_4\tau^2A\xi X$$
$$\lambda_2\tau_2+\tau\theta_2A^2\xi\lambda_1+\tau\theta_2A\xi\mu X+\tau\theta_2A^2\mu X\lambda_1+\tau\theta_2A\mu^2X^2)I_U-(\phi\tau_1d_2+\phi\tau_2\sigma_1+\tau^3\beta YX\pi)I_U-(\theta_1\tau^2+\theta_2A$$
$$+\lambda^2\tau^2+\lambda_1Ad_2d_3d_4\tau+\lambda_1Ad_2d_3\theta_2A)=0$$

Applying Descartes's Rule of Sign

The number of positive root is the number of sign change

$$aI_U^{*3}+bI_U^{*2}+cI_U^*+d=0$$

Where $a=d_1d_2d_3d_4\tau^3\beta XY^2\lambda_2\tau_2+d_1d_2d_3\tau^2\theta_2A\beta XY^2\lambda_2\tau_2$

$$b=d_1d_2d_3d_4\tau^2\xi\lambda_2\tau_2Y+d_1d_2d_3d_4\tau^4\mu X\lambda_2Y+d_1d_2d_3d_4\tau^3\beta XY\lambda_1A+d_1d_2d_3d_4\tau^3\beta X^2Y\mu+$$

$$c=d_1d_2d_3d_4\tau^2\xi\lambda_1A+d_1d_2d_3d_4\tau^2\xi\mu X+d_1d_2d_3d_4\tau^2\lambda_1XA+d_1d_2d_3d_4\tau^2X^2\mu+d_1d_2d_3d_4\tau^2\xi A^2X\lambda_1$$
$$d_1d_2d_3\tau\theta_2A\xi\lambda_2\tau_2Y+d_1d_2d_3\tau\theta_2A\mu XY\lambda_2\tau_2+d_1d_2d_3\tau^2\theta_2A^2\beta XY\lambda_1+d_1d_2d_3\tau^2\theta_2A\beta X^2Y\mu$$

$$+d_1d_2d_3d_4\tau^2\xi A\mu X^2+d_1d_2d_3d_4\tau^2\xi AX\lambda_2\tau_2+d_1d_2d_3\tau\theta_2A^2\xi\lambda_1+d_1d_2d_3\tau\theta_2A\xi\mu X+d_1d_2d_3\tau\theta_2A^2\mu X\lambda_1$$
$$+d_1d_2d_3\tau\theta_2A\mu^2X^2-(\phi\tau_1d_2+\phi\tau_2\sigma_1+\tau^3\beta XY\pi)$$
$$d=-(\theta_1\tau^2+\theta_2A+\lambda_2\tau^2+\lambda_1Ad_2d_3d_4\tau+\lambda_1Ad_2d_3\theta_2A)$$

With

$$C_1=d_1d_2d_3\tau(d_4\tau\xi\lambda_1A+d_4\tau\xi\mu X+d_4\tau\mu X\lambda_1A+d_4\tau\mu X^2+d_4\tau\xi A^2X\lambda_1+d_4\tau\xi A\mu X^2+d_4\tau\xi AX\lambda_2\tau_2+\theta_2A^2\xi\lambda_1+$$
$$\theta_2A\xi\mu X+\theta_2A^2\mu X\lambda_1+\theta_2A\mu^2X^2$$

And

$$C_2=-(\phi\tau_1d_2+\phi\tau_2\sigma_1+\tau^2\beta XY\pi)$$

Such that $C=C_1-C_2$

$If\ C_1\succ C_2, then\ C\succ 0,$ then there is only one sign change in the polynomial equation. Hence, there are two endemic equilibrium, the third is a negative equilibrium and is biologically not meaningful.

$If\ C_1\prec C_2, then\ C\prec 0,$ also there is only one sign change in polynomial equation.

Thus the model has two endemic equilibrium point and so there is possibility of backward bifurcation arising from the multiple equilibrium.

Local Stability of Disease Free Equilibrium (DFE) of Asthma Disease Model

Theorem 3.3 The disease free equilibrium point is locally asymptotically stable if $R_0<1$ and unstable if $R_0>1$. Then the theorem implies that the disease can be eliminated from the community

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Proof: To prove local stability of disease free equilibrium point, firstly the system was linearized and then Jacobian matrix was obtained at the disease free equilibrium point
given as;

$$J(S^0,0,00,0,0,0) = \begin{pmatrix} -\mu & -\frac{\beta\pi}{\mu} & 0 & 0 & 0 & \sigma_2\phi & 0 \\ 0 & -\theta_1-\theta-\mu & 0 & 0 & 0 & (1-\sigma_2)\phi & 0 \\ 0 & \frac{(1-\varepsilon_2)\beta\pi}{\mu} & -\mu & 0 & 0 & 0 & \frac{(1-\varepsilon_1)\gamma\pi}{\mu} \\ 0 & \frac{\varepsilon_2\beta\pi}{\mu}+\theta_1 & 0 & -(\sigma_1+\tau_1+\mu+\alpha) & 0 & 0 & \frac{\varepsilon_1\gamma\pi}{\mu} \\ 0 & 0 & 0 & \sigma_1 & -(\tau_2+\mu+\alpha) & 0 & 0 \\ 0 & 0 & 0 & \tau_1 & \tau_2 & -(\phi+\mu) & 0 \\ 0 & q & 0 & 0 & 0 & 0 & -\tau \end{pmatrix}$$

for simplicity we let; $K_1 = \frac{\beta\pi}{\mu}, K_2 = \theta_1+\theta+\mu, K_3 = \frac{(1-\varepsilon_2)\beta\pi}{\mu}, K_4 = \frac{\varepsilon_2\beta\pi}{\mu}+\theta_1,$

$K_5 = (\sigma_1+\tau_1+\mu+\alpha), K_6 = (\tau_2+\mu+\alpha), K_7 = (1-\sigma_2)\phi, K_8 = (\phi+\mu), K_9 = \frac{(1-\varepsilon_1)\gamma\pi}{\mu},$

$K_{10} = \frac{\varepsilon_1\gamma\pi}{\mu}$

upon the substitution of the above representation, we obtain the following;

$$J(E^0) = \begin{pmatrix} -\mu & -K_1 & 0 & 0 & 0 & \sigma_2\phi & 0 \\ 0 & -K_2 & 0 & 0 & 0 & K_7 & 0 \\ 0 & K_3 & -\mu & 0 & 0 & 0 & K_9 \\ 0 & K_4 & 0 & -K_5 & 0 & 0 & K_{10} \\ 0 & 0 & 0 & \sigma_1 & -K_6 & 0 & 0 \\ 0 & 0 & 0 & \tau_1 & \tau_2 & -K_8 & 0 \\ 0 & q & 0 & 0 & 0 & 0 & -\tau \end{pmatrix}$$

$$|J-\lambda I| = \begin{vmatrix} -\mu-\lambda & -K_1 & 0 & 0 & 0 & \sigma_2\phi & 0 \\ 0 & -K_2-\lambda & 0 & 0 & 0 & K_7 & 0 \\ 0 & K_3 & -\mu-\lambda & 0 & 0 & 0 & K_9 \\ 0 & K_4 & 0 & -K_5-\lambda & 0 & 0 & K_{10} \\ 0 & 0 & 0 & \sigma_1 & -K_6-\lambda & 0 & 0 \\ 0 & 0 & 0 & \tau_1 & \tau_2 & -K_8-\lambda & 0 \\ 0 & q & 0 & 0 & 0 & 0 & -\tau-\lambda \end{vmatrix} = 0$$

Expand along the first column to obtain

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$$(-\mu-\lambda)\begin{vmatrix} -K_2-\lambda & 0 & 0 & 0 & K_7 & 0 \\ K_3 & 0 & 0 & 0 & 0 & 0 \\ K_4 & -\mu-\lambda & -K_5-\lambda & 0 & 0 & K_9 \\ 0 & 0 & \sigma_1 & -K_6-\lambda & 0 & K_{10} \\ 0 & 0 & \tau_1 & \tau_2 & -K_8-\lambda & 0 \\ q & 0 & 0 & 0 & 0 & -\tau-\lambda \end{vmatrix} = 0 \qquad -(-\mu-\lambda)=0$$

or $$\begin{vmatrix} -K_2-\lambda & 0 & 0 & K_7 & 0 \\ K_4 & -K_5-\lambda & 0 & 0 & K_{10} \\ 0 & \sigma_1 & -K_6-\lambda & 0 & 0 \\ 0 & \tau_1 & \tau_2 & -K_8-\lambda & 0 \\ q & 0 & 0 & 0 & -\tau-\lambda \end{vmatrix} = 0$$

Therefore $\lambda_1 = -\mu$

$$\begin{vmatrix} -K_2-\lambda & 0 & 0 & 0 & K_7 & 0 \\ K_3 & -\mu-\lambda & 0 & 0 & 0 & 0 \\ K_4 & 0 & -K_5-\lambda & 0 & 0 & K_9 \\ 0 & 0 & \sigma_1 & -K_6-\lambda & 0 & K_{10} \\ 0 & 0 & \tau_1 & \tau_2 & -K_8-\lambda & 0 \\ q & 0 & 0 & 0 & 0 & -\tau-\lambda \end{vmatrix} = 0$$

also, expand along the second columns we obtain the following;

$$-(-\mu-\lambda)\begin{vmatrix} -K_2-\lambda & 0 & 0 & K_7 & 0 \\ K_4 & -K_5-\lambda & 0 & 0 & K_{10} \\ 0 & \sigma_1 & -K_6-\lambda & 0 & 0 \\ 0 & \tau_1 & \tau_2 & -K_8-\lambda & 0 \\ q & 0 & 0 & 0 & -\tau-\lambda \end{vmatrix} = 0$$

$-(-\mu-\lambda)=0$ or $$\begin{vmatrix} -K_2-\lambda & 0 & 0 & K_7 & 0 \\ K_4 & -K_5-\lambda & 0 & 0 & K_{10} \\ 0 & \sigma_1 & -K_6-\lambda & 0 & 0 \\ 0 & \tau_1 & \tau_2 & -K_8-\lambda & 0 \\ q & 0 & 0 & 0 & -\tau-\lambda \end{vmatrix} = 0$$

Therefore, $\lambda_2 = -\mu$

Similarly, expand this along the first row $$\begin{vmatrix} -K_2-\lambda & 0 & 0 & K_7 & 0 \\ K_4 & -K_5-\lambda & 0 & 0 & K_{10} \\ 0 & \sigma_1 & -K_6-\lambda & 0 & 0 \\ 0 & \tau_1 & \tau_2 & -K_8-\lambda & 0 \\ q & 0 & 0 & 0 & -\tau-\lambda \end{vmatrix} = 0$$

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$$(-K_2-\lambda)\begin{vmatrix} -K_5-\lambda & 0 & 0 & K_{10} \\ \sigma_1 & -K_6-\lambda & 0 & 0 \\ \tau_1 & \tau_2 & -K_8-\lambda & 0 \\ 0 & 0 & 0 & -\tau-\lambda \end{vmatrix} - K_7\begin{vmatrix} K_4 & -K_5-\lambda & 0 & K_{10} \\ 0 & \sigma_1 & -K_6-\lambda & 0 \\ 0 & \tau_1 & \tau_2 & 0 \\ q & 0 & 0 & -\tau-\lambda \end{vmatrix} = 0$$

$$(-K_2-\lambda)\left\{(-K_8-\lambda)\begin{vmatrix} -K_5-\lambda & 0 & K_{10} \\ \sigma_1 & -K_6-\lambda & 0 \\ 0 & 0 & -\tau-\lambda \end{vmatrix}\right\} - K_7\left\{K_4\begin{vmatrix} \sigma_1 & -K_6-\lambda & 0 \\ \tau_1 & \tau_2 & 0 \\ 0 & 0 & -\tau-\lambda \end{vmatrix} - q\begin{vmatrix} K_5-\lambda & 0 & K_{10} \\ \sigma_1 & -K_6-\lambda & 0 \\ \tau_1 & \tau_2 & 0 \end{vmatrix}\right\} = 0$$

after the expansion and simplification, we obtain the following characteristics of polynomial equation: $A_5\lambda^5 + A_4\lambda^4 + A_3\lambda^3 + A_2\lambda^2 + A_1\lambda + A_0 = 0$ (34)

Where $A_5 = 1$, $A_4 = K_2 + K_5 + \tau - K_8 + K_6$

, $A_3 = K_2K_5 + K_2\tau - K_2K_8 + K_2K_6 + K_5\tau - K_5K_8 + K_6K_5 - K_2\tau + K_6\tau - K_6K_8$

$A_2 = K_2K_5\tau_1 - K_2K_5K_8 + K_2K_5K_6 - K_2K_8\tau + K_2K_6\tau - K_2K_6K_8 + K_5K_6\tau - K_5K_8\tau - K_5K_6K_8 - K_6K_8\tau - K_4K_7\tau_1$

$A_1 = (K_2K_5K_6\tau - K_2K_5K_8\tau - K_2K_5K_6K_8 - K_2K_6K_8\tau - K_5K_6K_8\tau) - K_7(K_4\sigma_1\tau_2 + K_4K_6\tau\tau_1 + K_4K_6\tau_1 + K_{10}q\tau_1)$

$A_0 = K_2K_5K_6K_8\tau + K_7(K_4\tau\tau_2\sigma_1 - K_4\tau\tau_1K_6 + qK_{10}\tau_2\sigma_1 - qK_{10}K_6\tau_2\sigma_1)$

The Routh Hurwitz criterion was applied to determine the nature of the roots of the polynomial, which are positive, then all the eigenvalues of the Jacobian matrix have negative real roots when $R_0 < 1$, therefore, the disease free equilibrium is locally asymptotically stable

Global Stability of Disease Free Equilibrium (DFE) of Asthma Disease Model

This is prove through the comparison theorem

$$\begin{pmatrix} E' \\ I_U' \\ I_D' \\ P' \end{pmatrix} \le (F-V).\begin{pmatrix} E \\ I_U \\ I_D \\ P \end{pmatrix}$$ We now compute the eigenvalues of $F-V$

$$F-V = \begin{pmatrix} 0 & 0 & 0 & \frac{(1-\varepsilon_1)\xi\pi}{\mu} \\ 0 & 0 & 0 & \frac{\varepsilon_1\xi\pi}{\mu} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} - \begin{pmatrix} \mu & 0 & 0 & 0 \\ 0 & d_2 & 0 & 0 \\ 0 & -\sigma_1 & d_3 & 0 \\ -A & -A & -A & \tau-A \end{pmatrix}$$

then $F-V = \begin{pmatrix} -\mu & 0 & 0 & \frac{(1-\varepsilon_1)\xi\pi}{\mu} \\ 0 & -d_2 & 0 & \frac{\varepsilon_1\xi\pi}{\mu} \\ 0 & \sigma_1 & -d_3 & 0 \\ A & A & A & A-\tau \end{pmatrix}$ To find the eigenvalues of F-

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V, that is $|(F-V)-\lambda I|=0$

$$\begin{vmatrix} -(\mu+\lambda) & 0 & 0 & \frac{(1-\varepsilon_1)\xi\pi}{\mu} \\ 0 & -(d_2+\lambda) & 0 & \frac{\varepsilon_1\xi\pi}{\mu} \\ 0 & \sigma_1 & -(d_3+\lambda) & 0 \\ 0 & A & A & -(\tau-A+\lambda) \end{vmatrix}=0$$

$$-(\mu+\lambda)\begin{vmatrix} -(d_2+\lambda) & 0 & \frac{\varepsilon_1\xi\pi}{\mu} \\ \sigma_1 & -(d_3+\lambda) & 0 \\ A & A & -(\tau-A+\lambda) \end{vmatrix}\frac{-(1-\varepsilon_1)\xi\pi}{\mu}\begin{vmatrix} 0 & -(d_2+\lambda) & 0 \\ 0 & \sigma_1 & -(d_3+\lambda) \\ A & A & A \end{vmatrix}=0$$

$$(\mu+\lambda)(d_2+\lambda)(d_3+\lambda)(\tau-A+\lambda)-(\mu+\lambda)\frac{\varepsilon_1\xi\pi}{\mu}\left[(\sigma_1 A+(d_3+\lambda)A\right]-\frac{(1-\varepsilon_1)\xi\pi}{\mu}(d_2+\lambda)(0+(d_3+\lambda)A)=0$$

$$(\mu+\lambda)(d_2+\lambda)(\tau-A+\lambda)-\frac{(\mu+\lambda)\varepsilon_1\xi\pi\sigma_1 A}{\mu}-\frac{(\mu+\lambda)(d_3+\lambda)\varepsilon_1\xi\pi A}{\mu}-\frac{(1-\varepsilon_1)(d_2+\lambda)(d_3+\lambda)\xi\pi A}{\mu}=0$$

$$\lambda^4+(\mu+d_2+d_3+\tau-A)\lambda^3+\left[\mu d_2+(\mu+d_2)(d_3+\tau-A)+d_3(\tau-A)-\frac{\xi\pi A}{\mu}\right]\lambda^2+$$

$$\left[\mu d_2(d_3+\tau-A)+(\mu+d_2)(\tau-A)d_3-\frac{\varepsilon_1\xi\pi A}{\mu}(\sigma_1-(\mu+d_3))-\frac{(1-\varepsilon_1)(d_2+d_3)\xi\pi A}{\mu}\right]\lambda+$$

$$\mu d_2 d_3(\tau-A)\left[1-(\frac{\varepsilon_1\xi\pi\mu A(\sigma_1+d_3)+(1-\varepsilon_1)d_2 d_3\xi\pi A}{\mu^2 d_2 d_3(\tau-A)})\right]=0$$

$$\lambda^4+(\mu+d_2+d_3+\tau-A)\lambda^3+\left[\mu d_2+(\mu+d_2)(d_3+\tau-A)+d_3(\tau-A)-\frac{\xi\pi A}{\mu}\right]\lambda^2+$$

$$\mu d_2(d_3+\tau-A)+(\mu+d_2)(\tau-A)d_3\left[1-\frac{\xi\pi A}{\mu}(\frac{\varepsilon_1(\sigma_1-\mu-d_3)+(1-\varepsilon_1)(d_2+d_3)}{\mu d_2(d_3+\tau-A)+(\mu+d_2)(\tau-A)d_3}\right]\lambda+ \quad (35)$$

$$\mu d_2 d_3(\tau-A)(1-R_0)=0$$

Now if $\frac{\xi\pi A}{\mu}\prec \mu d_2+(\mu+d_2)(d_3+\tau+A)+d_3(\tau-A)$ then

$$\frac{\xi\pi A}{\mu}\left[\frac{\varepsilon_1(\sigma_1-\mu-d_3)+(1-\varepsilon_1)(d_2+d_3)}{\mu d_2(d_3+\tau-A)+(\mu+d_2)(\tau-A)d_3}\right]\prec 1 \text{ and } R_0<1$$

Then by Descartes rule of sign, the polynomial equation (35) has no sign change. This shows that all the eigenvalues are negative. Hence, the disease free equilibrium is globally asymptotically stable, if $R_0 \prec 1.$

Numerical Simulation Results

The analytical results of this study are illustrated by carrying out numerical simulations of the models using parameter values obtained from literature as shown in Table3 and the following results are obtained via MAPLE 18 software.

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Table 3. Parameters values used for the numerical simulation

Parameter	Symbol	Parameter Value	Source
Recruitment rate of Susceptible	π	100	Naresh and Tripathi (2009)
Recruitment rate of Smokers	Q	60	
Interaction of susceptibles with smokers	β	0.0002	Naresh and Tripathi (2009)
Interaction of susceptibles with pollutants	ξ	0.0003	Junehyuk et. al., (2017)
Natural death rate	μ	1/70 = 0.014	Naresh and Tripathi (2009)
Disease induced death rate	α	1 / 50 = 0.018	Naresh and Tripathi (2009)
Rate at which smokers quit smoking	θ	0.002	Chatzimichail et al.,(2013)
Cumulative density of pollutants	A	10	Kavitha (2016)
Rate at which pollution increase in smoker's smoke	Q	0.00001	Ana (2016)
Depletion rate of environmental discharge as environment cleans	τ	0.01	Junehyuk et. al., (2017)
Interaction of exposed persons with pollutants	λ_1	0.0001	Kavitha (2016)
Interaction of exposed persons with smokers	λ_2	0.00015	Kavitha (2016)
Rate at which smokers become infected	θ_1	0.0002	Shakirova et. al., (2013)
Rate at which smokers came into the contact of pollution	θ_2	0.00025	Kavitha (2016)

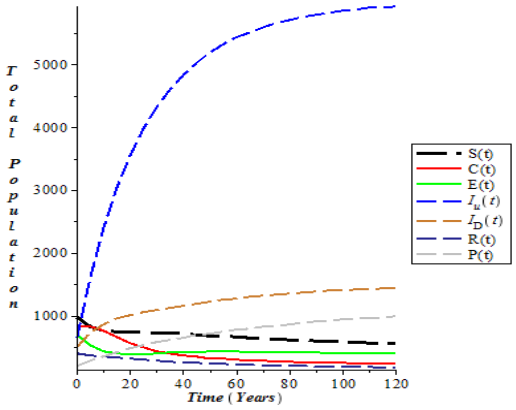

Figure.2. Shows the behavior of total population at different time

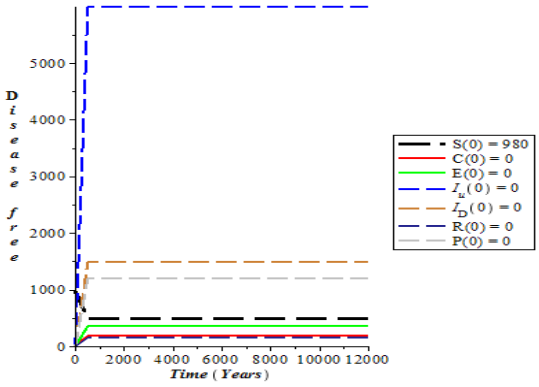

Figure.3. Graph of disease free equilibrium point of model at different time

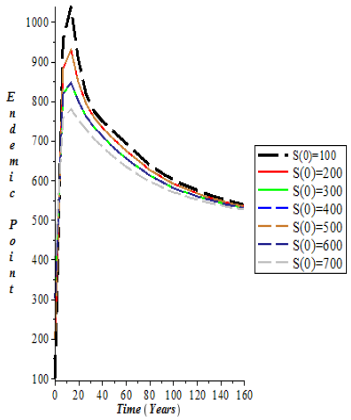

Figure.4. Graph of global stability of endemic equilibrium point of susceptible at different time

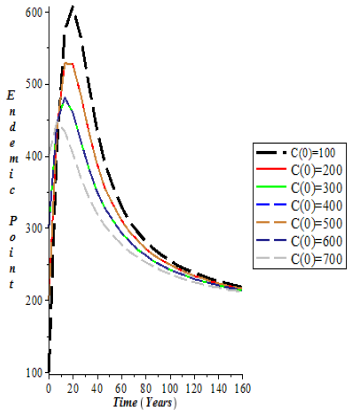

Figure.5. Graph of global stability of endemic equilibrium point of smokers at different time

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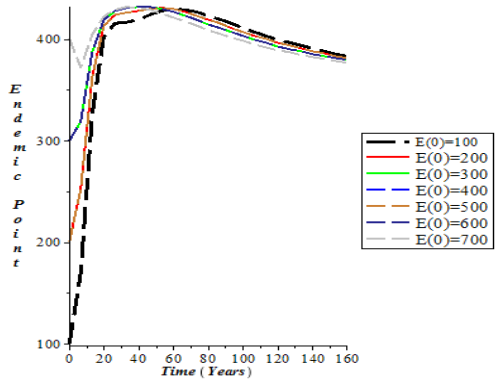

Figure.6. Graph of global stability of endemic equilibrium point of expose at different time

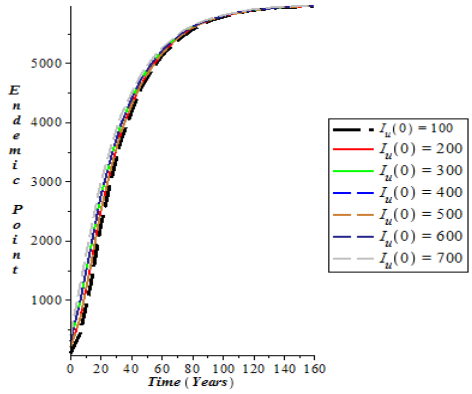

Figure.7. Graph of global stability of endemic equilibrium point of infected undetected at different time

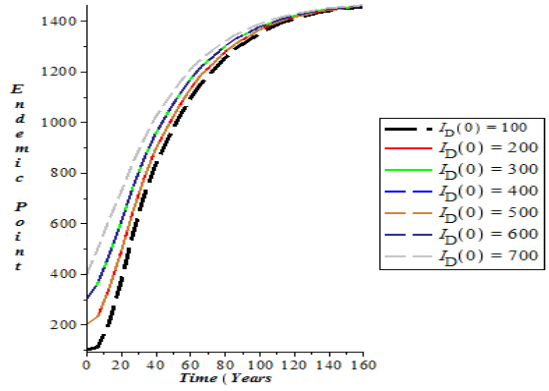

Figure.8. Graph of global stability of endemic equilibrium point of infected detected at different time

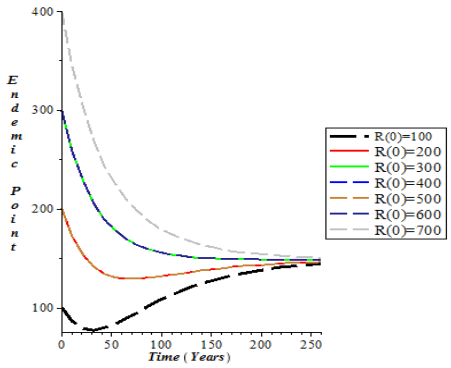

Figure.9. Graph of global stability of endemic equilibrium point of recovered at different time

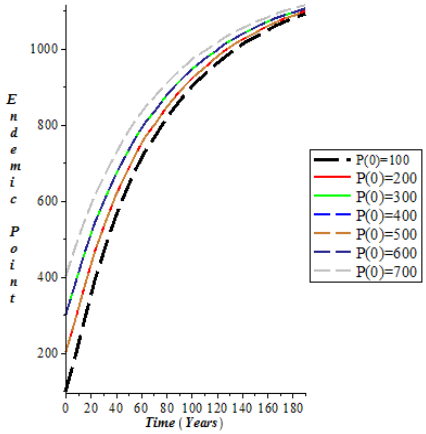

Figure.10. Graph of global stability of endemic equilibrium point of pollutant at different time

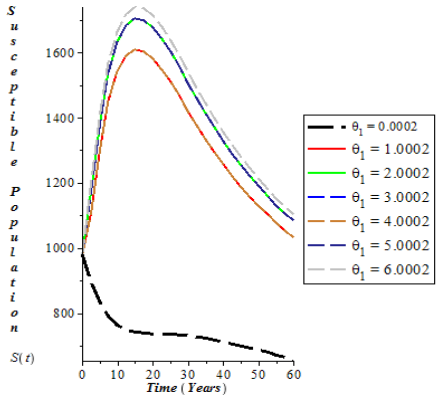

Figure.11. Impact of rate at which smokers become infected on susceptible at different time

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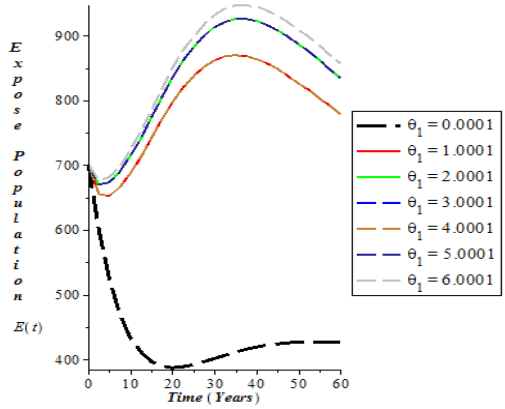

Figure.12. Impact of rate at which smokers become infected on expose at different time

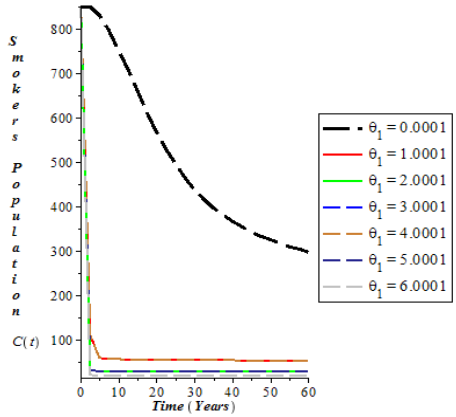

Figure.13. Impact of rate at which smokers become infected on smokers population at different time

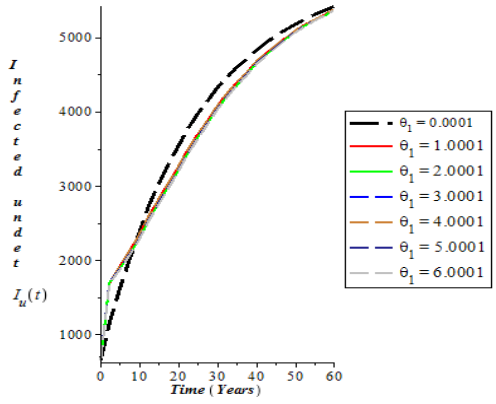

Figure.14. Impact of rate at which smokers become infected on infected undetected at different time

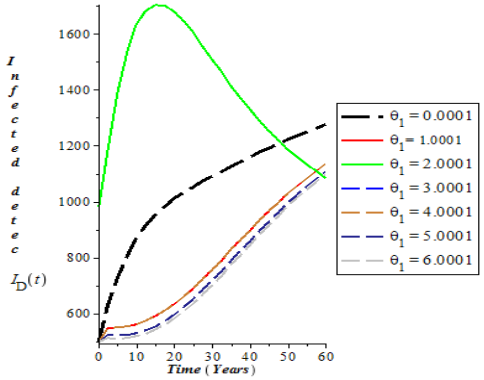

Figure.15. Impact of rate at which smokers become infected on infected detected at different time

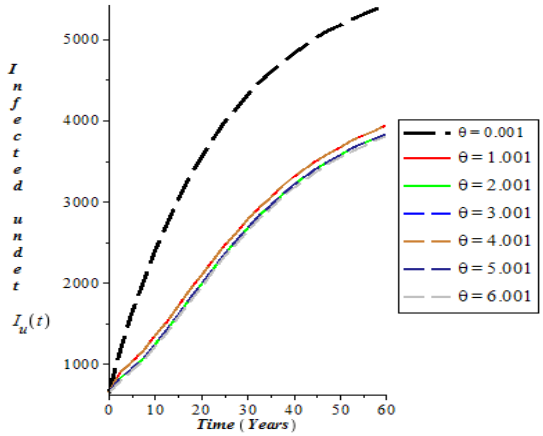

Figure.16. Impact of rate at which smoker quit on infected undetected at different time

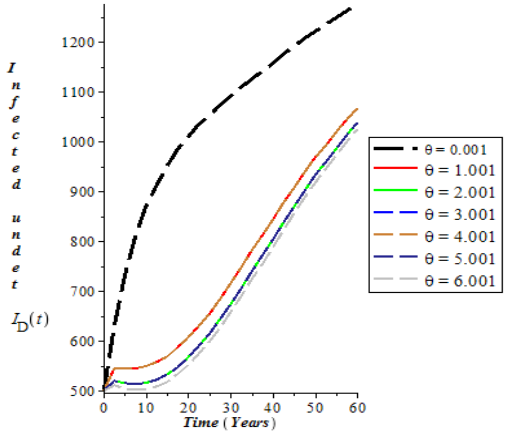

Figure.17. Impact of rate at which smoker quit on infected detected at different time

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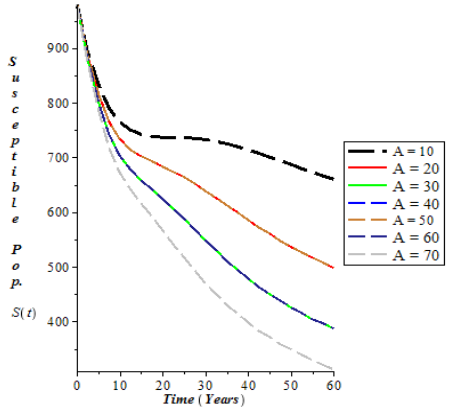

Figure.18. Impact of cumulative density of pollutants on susceptible at different time

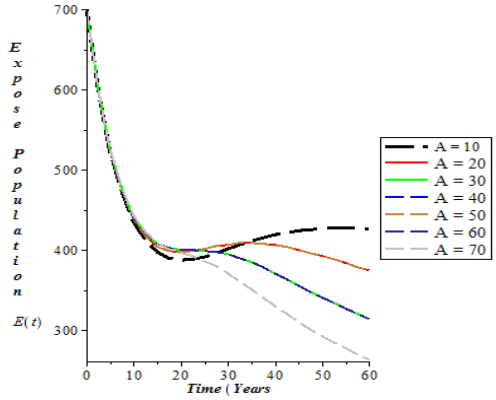

Figure.19. Impact of cumulative density of pollutants on expose at different time

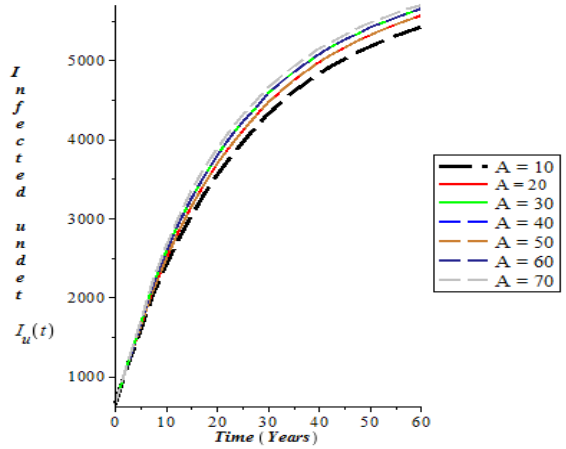

Figure.20. Impact of cumulative density of pollutants on infected undetected at different time

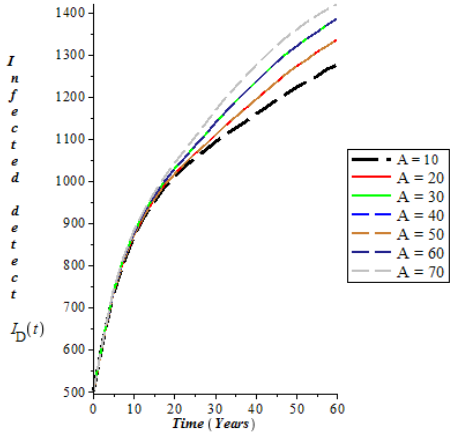

Figure .21. Impact of cumulative density of pollutants on infected detected at different time

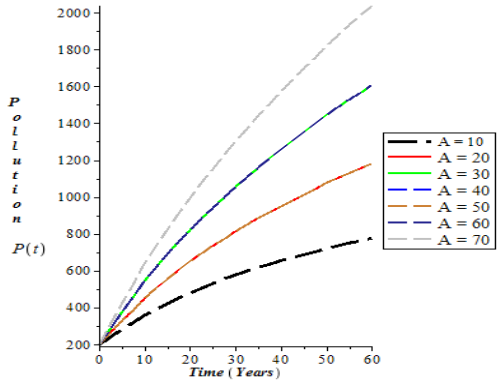

Figure .22. Impact of cumulative density of pollutants on pollution at different time

DISCUSSION

Figure.2, it was discovered that each classes or compartments behaves in different ways in a total population. While some increases (infected undetected, infected detected, environmental pollution), some decreases (susceptible, exposed, recovered, smokers).Fig.3, it was discovered that at the disease free equilibrium point, all compartment except susceptible were at the steady state and there is no disease. Fig.4, it was discovered that at the global stability of endemic equilibrium point, susceptible increase and at a point decreases. This means that due to the presence of disease in the population, the number in susceptible class decreases while others will move to other human compartments. Fig.5, it was discovered that at the global stability of endemic equilibrium point, smokers increase and at a point decreases. This means that some people in the smoker's class who are infected will move to another compartment. Fig.6, it was discovered that at the global stability of endemic equilibrium point, all human are expose to the disease due to the environmental pollution. Also, Fig.7, it was discovered that at the global stability of endemic equilibrium point, infected undetected at different time increases. This means that the number of infected undetected increases due to the presence of disease in the environment and these people did not do medical check-up.

Fig.8, it was discovered that the global stability of endemic equilibrium point, infected detected at different time increases. It means that the total number of infected detected increases due to

the presence of disease in the environment and thereby needs proper and effective management of the disease. Fig.9, it was discovered that at the global stability of endemic equilibrium point, the number of people that recovered increases after taking necessary measurement. This means that the number of people in recovered class increases after taking proper and effective measures. Fig.10, it was discovered that at the global stability of endemic equilibrium point, the environmental pollution class dictate the tune of the presence of the disease in the environment. If the pollution increases, there is a possibility to an increase in the disease and if it decreases, there is a possibility to a decrease of the disease in the environment. This means that there is need for proper control of pollution in the environment in other to reduce the environmental pollution and thereby reduce the possibility of having more disease in the population.

Fig.11, it was discovered that the rate at which smokers become infected on susceptible at different time increases thereby reduces the total number of population in susceptible class. This means that the impact of rate at which the smokers infected on susceptible increases, thereby affect the entire population. Fig.12, it was discovered that the rate at which smokers become infected on exposed class reduces. This means that the smokers who are already infected will move to the infected class and not exposed class again. Fig.13, it was discovered that the impact of rate at which smokers become infected initially higher and later smokers population reduces.

Fig. 14, it was discovered that the impact of rate at which smokers become infected on infected undetected increases as the year increases. In Fig.15, it was discovered that the impact of rate at which smokers become infected on infected detected at different time varies: The rate on susceptible class, smokers class, and infected undetected class continues to increase as the pollution class increases but the exposed class initially increases but later decreases. In Fig.16, it was discovered that the impact of rate at which smoker quit on infected undetected at different time on susceptible class, smokers class, exposed class, infected undetected class continue to increases. In Fig.17, it was discovered that the impact of rate at which smoker quit on infected detected increases in all classes as the time increases. Fig.18, it was discovered that the impact of cumulative density of pollutants on susceptible continues to decrease as the time increases. In Fig.19, it was discovered that the impact of cumulative density of pollutants on expose continues to decrease as the time increases and at a point only susceptible increase while other classes continue to decrease. In Fig.20, it was discovered that the impact of cumulative density of pollutant on infected undetected continue to increases over the years. Finally, in Fig.21, it was discovered that the impact of cumulative density of pollutants on infected detected continue to increase over the years. In Fig.22, it was discovered that the impact of cumulative density of pollutants increases over the years at different rates.

Conclusion

In this work, an improved model of asthma which includes infected undetected class and recovered (relieved) class was investigated. The model exhibits two equilibrium, namely disease free equilibrium and endemic equilibrium. The analysis showed that the model is unique, exist and well posed. It is assumes that susceptible become infected undetected when they are continuously exposed to smoking or when they enter into polluted environment and inhale the pollutants from the atmosphere. It has been shown in simulation that when pollutants are discharged into the environment at a constant rate and smokers are also enlisted into the population; the asthma disease persistence is higher, reason being that the interaction rate of susceptible with pollutants and with smokers' increases, the fractions of infected undetected increases as well. Finally, the spread of asthma can be control by restricting the smokers from the population and the rate of release of pollutants i s also controlled in the environment.

Conflict of interest: The authors declare no conflict of interest.

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