Available online at http://scik.org
Commun. Math. Biol. Neurosci. 2022, 2022:75
https://doi.org/10.28919/cmbn/7564
ISSN: 2052-2541

ECO-EPIDEMIOLOGICAL MODEL AND STABILITY ANALYSIS OF COTTON LEAF CURL VIRUS (CLCUV) TRANSMISSION DYNAMICS

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Abstract. In this paper, we develop a mathematical model for the transmission dynamics of Cotton leaf curl virus (CLCuV) disease in cotton. The models took into account both cotton and vector populations. Cotton populations are classified as susceptible (A) and infected (B). The vector population was further classified as susceptible (X) and infected (Y). We demonstrated that all model solutions are positive and bounded with initial circumstances from a specific meaningful set. The presence of unique CLCuV free and endemic equilibrium points is explored, and the basic reproduction number is calculated using the next generation matrix approach. The conditions for these equilibrium points' local and global asymptotic stability are then established. When the basic reproduction number is less than one, the system has a locally and globally asymptotically stable CLCuV free equilibrium point, and when the basic reproduction number is more than one, the system has a locally and globally asymptotically stable endemic equilibrium point. The simulation result agrees with the analytical results.

Keywords: eco-epidemiological model; cotton leaf curl virus; basic reproduction number; stability analysis.

2010 AMS Subject Classification: 92B05.

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Received June 21, 2022

1. INTRODUCTION

Cotton was first mentioned in Amharic word 'tet,' about 350 A.D., during the reign of King Aizana of Axum [1]. Ethiopians have farmed and utilized cotton since ancient times. Cotton (Gossypium spp.) is the world's most important fiber, oil, and protein producing crop [2]. Cotton, a one-of-a-kind fiber crop plant with a thousand faces, is known for its versatility, performance, appearance, natural comfort, and, above all, its numerous applications, which include astronaut in-flight space suits, towels, tarpaulins, tents, sheets, and all forms of clothing Vanitha et al. [3]. It is known as "white gold," is one of the most important cash crops in developing countries and exported to various nations as a raw material for indigenous textile and oil industries Sain et al. [4]. Cotton's significance is underscored by the fact that it is not only the world's most important fiber crop, but also the world's second-largest oilseed crop Zhang et al. [5].

Cotton is the most widely grown food and fiber crop in the world. Ethiopia is a cotton-producing and exporting country in Sub-Saharan Africa. It has a long history of cotton production, with an estimated area suitable for cultivation of roughly 2.6 million hectares. Ethiopian cotton is mostly exported to Africa, Asia, and Europe, with Asia accounting for 67% of total exports. Ethiopian cotton is now priced by the Textile Industry Development Institute Zeleke et al. [6]. The leaf is the most susceptible to diseases, which can cause plant damage and death. The majority of infections only damage the cotton plant's leaf parts Kumar et al. [7]. Cotton Leaf Curl Virus (CLCuV) illness is a major impediment to cotton production. It is caused by a genus of viruses known as Begomovirus, which is spread by whiteflies and poses a serious threat to the cotton crop Farooq et al. [8]. It can be seen in Africa, Pakistan, and northwestern India Sattar et al. [9]. Cotton symptoms appear 2-3 weeks after B. tabaci inoculation (discovered experimentally) and are marked by deep downward cupping of the youngest leaves [10]. Roguing, or the removal of infected plants, particularly ratoon cotton from the previous season's crop, is advised, but it appears to have little influence on the disease's spread. Cotton cultivars with resistance to herbicides and pesticides have recently been introduced.

Mathematicians and biologists have developed multiple ecoepidemiological models in recent decades to explore the spread of various infectious diseases [11, 12, 13, 14] and etc. Fouda et

al. [15] created a mathematical model of bleached cotton plain single jersey knitted fabrics that may be used to anticipate fabric attributes and define fabric geometrical relationships before manufacturing. A practical verification is performed at various cotton yarn counts and twist factors. Furthermore, the fabric measuring method measures the real yarn diameter and estimates the fabric thickness as a result. According to the findings, the thickness of a simple single jersey is related to three times the yarn diameter.

Levins et al. [16] describe the differential equations of prey-predator, crop-pest, and migratory effect interactions in their model. The relationship between a mathematical model and ecological stability was demonstrated using biological properties. The models were used to provide answers to environmental questions.

Hern'andez-Bautista et al. [17] proposed a mathematical model of cotton dyeing in cones on three scales: micro, meso, and macro. The parameters of the adsorption isotherms and independent variables of the Curcuma longa cotton dyeing process were determined using bibliographic data. To simulate cotton dyeing, mass and momentum conservation equations were applied. Banks et al. [18] developed a mathematical model and statistical models for making the optimal judgments, such as ANOVA-based model comparison tests and residual plot analysis. They also investigate the statistical assumptions that are typically made arbitrarily throughout the parameter estimate process, as well as the repercussions of making incorrect assumptions.

Mamatov et al. [19] presented a single parabolic-type boundary value issue for estimating the temperature field of raw cotton and air components in drum dryers . With the experimental data, a comparison analysis is conducted. The suggested model and numerical technique are shown to accurately explain the raw cotton drying process. Dome et al. [20] devised a mathematical model for estimating input demand in relation to cotton production costs. They also claim that cotton farmers are extremely susceptible to fluctuations in input prices, which influence whether they produce profitable or loss-making production when compared to total costs per hectare.

[21] provided six non-linear growth models for India Cotton area, production, and productivity statistics from 1980 to 2013: Monomolecular, Logistic, Gompertz, Richards, Quadratic, and Reciprocal growth. The selected model was chosen because it had the highest R2, Lower Residual Sum of Square, and Mean Square Error among the six models under consideration.

Aboukarima et al. [22] develop a multiple regression model to predict the leaf area of a cotton crop for use in agricultural studies. The created model may be a practical and quick alternative, particularly in locations where contemporary equipment or other instruments for measuring leaf area are unavailable.

Su et al. [23] explore the leaf area index (LAI) models and interactions between LAI, dry matter, and yield for cotton cultivated under three soil conditioners in Korla, Xinjiang, China, with the goal of improving water use efficiency and finding optimal soil conditioner application rates.

Khan et al. [24] investigate the association between cotton leaf curl virus (CLCuV) incidence, environmental variables, and silverleaf whitefly population in Pakistan's agricultural system. The mathematical relationship discovered can anticipate disease incidence in future months, which can aid agriculturists in disease control in Pakistan's agricultural areas.

Ahmad et al. [25] developed a mathematical model of cotton leaf curl virus infection in Pakistan, as well as its link to weather variables. They employ mathematics to connect the severity of the cotton leaf curl virus (CLCuV) to environmental factors including temperature, rainfall, and humidity, as well as the population of whiteflies in Pakistan's agricultural sector. Humidity and rainfall were discovered to be associated with the condition.

Cotton leaf curl virus, fiber quality, and yield components in germplasm imported from the United States were investigated by Saeed et al. [26]. 79 cotton genotypes were evaluated using statistical processes such as correlation analysis, clustering, and principal components. Cotton leaf curl virus demonstrated a substantial negative association with plant height, monopodial and sympodial branches, and a significant positive relationship with fiber fineness, but no relationship with other characteristics.

Motivated by the works of [23, 25, 26] we provide a new ecoepidemiological model that uses a ordinary differential equations to examine and analyze the dynamics of cotton leaf curl virus (CLCuV) in cotton plant populations. Furthermore, our current model is unique in that it divides the cotton leaf curl virus (CLCuV) model into two populations; cotton and vectors. Cotton and vector populations both have susceptible and infected individuals. We believe that the findings of our study will be valuable in identifying appropriate methods for preventing or eliminating

disease transmission. This research is organized as follows. In section two, a new mathematical model for the transmission dynamics of cotton leaf curl virus (CLCuV) is developed. Part three examines the presence and stability of cotton leaf curl virus (CLCuV) equilibria, as well as the positivity and boundedness of solutions. Part four is about numerical simulation. Finally, conclusions are given in part five.

2. Model Formulation

We divided the cotton leaf curl virus (CLCuV) model into cotton and vector populations in this part. There are susceptible and infected subgroups in these populations. A represents susceptible cotton, and B represents diseased cotton. Similarly, X represents a vulnerable vector and Y represents an infected vector. The model took into account the recruitment rate of susceptible vectors b_2 and their movement to infected vectors Y with θ_2 rate after consuming ill plants or cotton. The susceptible cotton A also replanted at rate b_1, and the illnesses spread to cotton when infected vectors Y eat susceptible cotton A at rate θ_1. Cotton that has been infected will never heal and will provide a very poor yield of cotton. The model also assumes that μ is the natural death rate for cotton population, and δ is natural death rate for vector population. In the model, ω is induced death rate due to disease. Moreover, the description of all parameters are given in table 1.

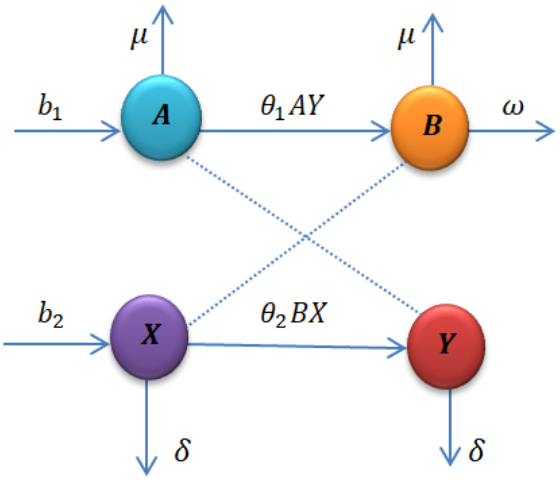

Figure 1. Flow chart of the model.

TABLE 1. Parameters of the model

Parameter	Description
b_1	Replanting rate of cotton.
b_2	Recruitment rate of vector.
θ_1	Infection rate of cotton.
θ_2	Infection rate of vector.
μ	Natural death rate of cotton.
δ	Natural death rate of vector.
ω	Induced death rate of cotton due to disease.

We can derive the following from the model's assumptions and flow chart in figure 1.

$$\begin{aligned} \frac{dA}{dt} &= b_1 - \theta_1 AY - \mu A, \\ \frac{dB}{dt} &= \theta_1 AY - (\mu + \omega)B, \\ \frac{dX}{dt} &= b_2 - \theta_2 BX - \delta X, \\ \frac{dY}{dt} &= \theta_2 BX - \delta Y, \end{aligned} \tag{1}$$

with

$$\begin{aligned} A(0) = A_0 \geq 0, B(0) = B_0 \geq 0, \\ X(0) = X_0 \geq 0, Y(0) = Y_0 \geq 0. \end{aligned} \tag{2}$$

3. MODEL ANALYSIS

3.1. Positivity of Solution. In this subsection, our model equation (1) to be ecoepidemiologically meaningful and well posed, it is necessary to prove that all state variables of system with positive initial data will remain positive for all times $t \geq 0$. The following theorem is used to demonstrate the positivity of the system of equation (1).

Theorem 1: Let $\Omega = (A,B,X,Y) \in \mathbf{R}^4 : A(0) > 0, B(0) > 0, X(0) >, Y(0) > 0$. Then the solution set $\{A(t), B(t), X(t), Y(t)\}$ of system of equation (1) is positive for all $t \geq 0$.

Proof: From the first equation of system equation (1), we have that:

$$\frac{dA}{dt} = b_1 - (\theta_1 Y + \mu)A \geq -(\theta_1 Y + \mu)A \tag{3}$$

Using separation variables and by integrating equation (3)

$$\int \frac{dA}{A} \geq -\int (\theta_1 Y + \mu)dt,$$

$$lnA(t) \geq -\int (\theta_1 Y + \mu)dt + c_1,$$

$$A(t) \geq e^{-\int (\theta_1 Y + \mu)dt + c_1},$$

$$A(t) \geq A(0)e^{-\int (\theta_1 Y + \mu)dt} \geq 0. \tag{4}$$

From the second equation of system equation (1), we have that:

$$\frac{dB}{dt} = \theta_1 AY - (\mu + \omega)B \geq -(\mu + \omega)B \tag{5}$$

Using separation variables and by integrating equation (5)

$$\int \frac{dB}{B} \geq -\int (\mu + \omega)dt,$$

$$lnB(t) \geq -(\mu + \omega)t + c_2,$$

$$B(t) \geq e^{-(\mu+\omega)t + c_2},$$

$$B(t) \geq B(0)e^{-(\mu+\omega)t} \geq 0. \tag{6}$$

Furthermore, using similar procedure on the above, we have

$$X(t) \geq X(0)e^{-\int (\theta_2 B + \delta)dt} \geq 0, \tag{7}$$

$$Y(t) \geq Y(0)e^{-\delta t} \geq 0. \tag{8}$$

Hence, all the solution sets are positive for $t \geq 0$, that is the model is meaningful and well posed.

3.2. Invariant Region. We determine a region in which the solution of system of equation (1) is bounded. Now, differentiating the total Cotton population $N_c = A + B$ with respect to time, we have

$$\frac{dN_c}{dt} = b_1 - \omega B - \mu N_c. \tag{9}$$

In the abscence of the death rate of cotton due to infection $\omega = 0$, equation (9) becomes

$$\frac{dN_c}{dt} \leq b_1 - \mu N_c \tag{10}$$

By re-arrranging and multiplying by integrating factor $e^{\int \mu dt} = e^{\mu t}$, we have

$$\frac{dN_c}{dt} e^{\mu t} + \mu N_c e^{\mu t} \leq b_1 e^{\mu t}. \tag{11}$$

That is

$$\frac{d}{dt}\left(N_c e^{\mu t}\right) \leq b_1 e^{\mu t}. \tag{12}$$

By integrating and solving equation (12), we obtain

$$N_c(t) \leq \frac{b_1}{\mu} + e^{-\mu t}\left(N_c(0) - \frac{b_1}{\mu}\right). \tag{13}$$

Taking the limit as $t \to \infty$ to the equation (13), we obtain

$$\Omega_c = \left\{(A,B) \in R_+^2 : N_c \leq \frac{b_1}{\mu}\right\} \tag{14}$$

And differentiating the cotton leaf curl virus population $N_v = X + Y$, we have

$$\frac{dN_v}{dt} = b_2 - \delta N_v. \tag{15}$$

Using similar procedure on the above

$$N_v(t) \leq \frac{b_2}{\delta} + e^{-\delta t}\left(N_v(0) - \frac{b_2}{\delta}\right). \tag{16}$$

Taking the limit as $t \to \infty$ to the equation (16), we obtain

$$\Omega_v = \left\{(X,Y) \in R_+^2 : N_v \leq \frac{b_2}{\delta}\right\} \tag{17}$$

As a result, the feasible solution set for the CLCuV model given by:

$$\Omega = \Omega_c \times \Omega_v = \left\{(A,B,X,Y) \in R_+^4 : N_c \leq \frac{b_1}{\mu} : N_v \leq \frac{b_2}{\delta}\right\} \tag{18}$$

is positively invariant, inside which the model is considered to be ecoepidemiologically meaningful and mathematically well posed.

3.3. Disease Free Equilibrium Point of the Model. The model's disease-free equilibrium points, E_0, are stationary solutions in which there is no infection. It is obtained by equating equation (1) to zero and using $B = 0$ and $Y = 0$. Then, disease free equilibrium point, E_0 of our model equation (1)is given by:

$$E_0 = (A^0, B^0, X^0, Y^0) = \left(\frac{b_1}{\mu}, 0, \frac{b_2}{\delta}, 0\right). \tag{19}$$

The basic reproduction number R_0: The basic reproduction number R_0 is the estimated number of secondary infections from a single newly infected individual delivered directly into a susceptible group, according to Kinene et al. [27]. It can be found by rewriting the system of equation (1) starting with newly infective classes and using the next generation matrix method:

$$\begin{aligned} \frac{dB}{dt} &= \theta_1 AY - (\mu + \omega)B, \\ \frac{dY}{dt} &= \theta_2 BX - \delta Y. \end{aligned} \tag{20}$$

Then, we consider

$$\boldsymbol{f} = \begin{pmatrix} \theta_1 AY \\ \theta_2 BX \end{pmatrix}, \boldsymbol{v} = \begin{pmatrix} (\mu + \omega)B \\ \delta Y \end{pmatrix} \tag{21}$$

Now, the jacobian matrix of $\boldsymbol{f}$ and $\boldsymbol{v}$ with respect to B and Y at disease free equilibrium point, $E_0 = \left(\frac{b_1}{\mu}, 0, \frac{b_2}{\delta}, 0\right)$ is:

$$\mathrm{F} = \begin{pmatrix} 0 & \frac{\theta_1 b_1}{\mu} \\ \frac{\theta_2 b_2}{\delta} & 0 \end{pmatrix},$$

$$\mathrm{V} = \begin{pmatrix} (\mu + \omega) & 0 \\ 0 & \delta \end{pmatrix} \tag{22}$$

Then, by the principle of next generation matrix, basic reproduction number R_0 is the dominant eigen value of the FV^{-1} or spectral radius of FV^{-1} where

$$FV^{-1} = \begin{pmatrix} 0 & \frac{\theta_1 b_1}{\mu} \\ \frac{\theta_2 b_2}{\delta} & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{(\mu+\omega)} & 0 \\ 0 & \frac{1}{\delta} \end{pmatrix}$$

$$= \begin{pmatrix} 0 & \frac{\theta_1 b_1}{\mu\delta} \\ \frac{\theta_2 b_2}{\delta(\mu+\omega)} & 0 \end{pmatrix} \tag{23}$$

The charactersic equations of equation (23) becomes;

$$\lambda^2 - \frac{b_1 b_2 \theta_1 \theta_2}{\mu\delta^2(\mu+\omega)} = 0. \tag{24}$$

That is

$$\lambda = \pm\sqrt{\frac{b_1 b_2 \theta_1 \theta_2}{\mu\delta^2(\mu+\omega)}}. \tag{25}$$

Since, basic reproductioin number R_0 is the maximum eigen values of FV^{-1} or the spectral radius of FV^{-1}. As a result,

$$R_0 = \sqrt{\frac{b_1 b_2 \theta_1 \theta_2}{\mu\delta^2(\mu+\omega)}}. \tag{26}$$

The two generation are required to transmission of CLCuV to take place in the cottton field, that is from an infectious cotton plant to susceptible vector and then from an infectious vector to susceptible cotton [28]. That is why the square root found in R_0. It implies that

$$\begin{aligned} R_0 &= \sqrt{\frac{b_1 b_2 \theta_1 \theta_2}{\mu\delta^2(\mu+\omega)}} = \sqrt{\frac{b_1\theta_1}{\mu(\mu+\omega)}\frac{b_2\theta_2}{\delta^2}} \\ &= \sqrt{\frac{b_1\theta_1}{\mu(\mu+\omega)}}\sqrt{\frac{b_2\theta_2}{\delta^2}} = R_{0c} \times R_{0v}. \end{aligned} \tag{27}$$

where $R_{0c} = \sqrt{\frac{b_1\theta_1}{\mu(\mu+\omega)}}$ is the cotton plants contribution when they infect the vector and $R_{0v} = \sqrt{\frac{b_2\theta_2}{\delta^2}}$ is the contribution of the vector population when it infects cotton plants.

3.4. Local Stability of the Disease Free Equilibrium Point. The linearization system of equation (1) at E_0 can be used to find the local stability of the model at disease-free equilibrium point, E_0.

Theorem 2: Disease free equilibrium point, E_0 of system of equation (1) is locally asymptotically stable, if $R_0 < 1$.

Proof. The Jacobian matrix of system of equation (1) is

$$J = \begin{pmatrix} -(\theta_1 Y + \mu) & 0 & 0 & -\theta_1 A \\ \theta_1 Y & -(\mu+\omega) & 0 & \theta_1 A \\ 0 & -\theta_2 X & -(\theta_2 B + \delta) & 0 \\ 0 & \theta_2 X & \theta_2 B & -\delta \end{pmatrix} \tag{28}$$

Evaluating the Jacobian matrix of system of equation (28) at disease free equilibrium point $E_0 = \left(\frac{b_1}{\mu}, 0, \frac{b_2}{\delta}, 0\right)$ is

$$J(E_0) = \begin{pmatrix} -\mu & 0 & 0 & -\frac{\theta_1 b_1}{\mu} \\ 0 & -(\mu+\omega) & 0 & \frac{\theta_1 b_1}{\mu} \\ 0 & -\frac{\theta_2 b_2}{\delta} & -\delta & 0 \\ 0 & \frac{\theta_2 b_2}{\delta} & 0 & -\delta \end{pmatrix} \tag{29}$$

The characteristic equation of Jacobian matrix of equation (29) at disease free equilibrium point, E_0 is $|J(E_0) - \lambda I_4| = 0$. That is

$$\begin{vmatrix} -\mu-\lambda & 0 & 0 & -\frac{\theta_1 b_1}{\mu} \\ 0 & -(\mu+\omega)-\lambda & 0 & \frac{\theta_1 b_1}{\mu} \\ 0 & -\frac{\theta_2 b_2}{\delta} & -\delta-\lambda & 0 \\ 0 & \frac{\theta_2 b_2}{\delta} & 0 & -\delta-\lambda \end{vmatrix} = 0 \tag{30}$$

Evaluating equation (30) simplifying it, we get

$$(-\mu-\lambda)(-\delta-\lambda)(\lambda^2 + d_1\lambda + d_2) = 0. \tag{31}$$

where

$$\begin{aligned} d_1 &= \mu + \omega + \delta, \\ d_2 &= (\mu+\omega)\delta - \frac{b_1 b_2 \theta_1 \theta_2}{\mu\delta} = (\mu+\omega)\delta\left[1 - \frac{b_1 b_2 \theta_1 \theta_2}{\mu\delta^2(\mu+\omega)}\right] \\ &= (\mu+\omega)\delta(1 - R_0^2). \end{aligned} \tag{32}$$

Clearly,from equation (31),we observe that

$$\lambda_1 = -\mu < 0,$$

$$\lambda_2 = -\delta < 0, \tag{33}$$

and from the last expression of equation (31) , that is

$$\lambda^2 + d_1\lambda + d_2 = 0 \tag{34}$$

by using the Routh-Hurwitz criteria, equation (34) has strictly negative real root if $d_1 > 0$ and $d_2 > 0$. Clearly, we observe that $d_1 = \mu + \omega + \delta > 0$ and

$$d_2 = (\mu + \omega)\delta(1 - R_0^2) > 0 \tag{35}$$

if $(1 - R_0^2) > 0$. That is $R_0^2 < 1$ implies that $R_0 < 1$. As a result, our model equation (1) at E_0 offers all eigenvalues with a negative real part, and so it is locally asymptotically stable if $R_0 < 1$.

3.5. Global Stability of the Disease Free Equilibrium Point. To establish the global stability of the disease free equilibrium point E_0, we use the method proposed by Castillo-Chavez et al. [29]. Based on Castillo-Chavez et al. [29], we have written the system of equation (1) in the following form:

$$\begin{aligned} \frac{dM}{dt} &= J(M,L), \\ \frac{dL}{dt} &= P(M,L), \\ P(M,0) &= 0, \end{aligned} \tag{36}$$

where $M = (A,X) \in R^2$ represent the number of uninfected classes, while, $L = (B,Y) \in R^2$ represent the number of infected classes and $E_0 = (M^*,0)$ represents the disease-free equilibrium of this system. The disease-free equilibrium E_0 is globally asymptotically stable equilibrium for the model if the following conditions are fulfilled:

(1) $\frac{dM}{dt} = J(M,0), M^*$ is globally asymptotically stable.
(2) $\frac{dL}{dt} = D_L P(M^*,0)L - \hat{P}(M,L), \hat{P}(M,L) \geq 0 \; \forall (M,L) \in \Omega$.

where $D_L P(M^*,0)$ is an M-matrix and $P(M,L)$ taken in (B,Y) and evaluated at $(M^*,0) = \left(\frac{b_1}{\mu},\frac{b_2}{\delta},0,0\right)$. If system of equation(24) satisfes the above conditions, then the following theorem holds.

Theorem 3: The disease free equilibrium point, $E_0 = (M^*,0)$ of system of equation (36) is globally asymptotically stable if $R_0 \leq 1$ and conditions (1) and (2) are holds.

Proof: From our model of equation (1), we can obtain $J(M,L)$ and $P(M,L)$:

$$J(M,L) = \begin{pmatrix} b_1 - \theta_1 AY - \mu A \\ b_2 - \theta_2 BX - \delta X \end{pmatrix}$$

$$P(M,L) = \begin{pmatrix} \theta_1 AY - (\mu+\omega)B \\ \theta_2 BX - \delta Y \end{pmatrix}$$

Now, we consider the reduced system $\frac{dM}{dt} = J(M,0)$ from condition (1)

$$\frac{dA}{dt} = b_1 - \mu A, \tag{37}$$

$$\frac{dX}{dt} = b_2 - \delta X. \tag{38}$$

$M^* = \left(\frac{b_1}{\mu},\frac{b_2}{\delta}\right)$ is a globally asymptotically stable equilibrium point for the reduced system $\frac{dM}{dt} = J(M,0)$. This can be verifed from the solution of the equation (37) ; we get $A(t) = \frac{b_1}{\mu} + \left(A(0) - \frac{b_1}{\mu}\right)e^{-\mu t}$ which approaches $\frac{b_1}{\mu}$ as $t \to \infty$ and from equation (38), we obtain $X(t) = \frac{b_2}{\delta} + (X(0) - \frac{b_2}{\delta})e^{-\delta t}$ which approaches $\frac{b_2}{\delta}$ as $t \to \infty$, We note that this asymptomatic dynamics is independent of the initial conditions in Ω; therefore the convergence of the solutions of the reduced system (37) and (38) is global in Ω. Now we compute

$$D_L P(M^*,0) = \begin{pmatrix} -(\mu+\omega) & \frac{b_1\theta_1}{\mu} \\ \frac{b_2\theta_2}{\delta} & -\delta \end{pmatrix} \tag{39}$$

Then, $P(M,L)$ can be written as

$$P(M,L) = D_L P(M^*,0)L - \hat{P}(M,L) \tag{40}$$

and we want to show $\hat{P}(M,L)$,which is obtained as

$$\hat{P}(M,L) = \begin{pmatrix} \theta_1 Y\left(\frac{b_1}{\mu} - A\right) \\ \theta_2 B\left(\frac{b_2}{\delta} - X\right) \end{pmatrix} \tag{41}$$

Here $\frac{b_1}{\mu} \geq A$ and $\frac{b_2}{\delta} \geq B$. Hence it is clear that $\hat{P}(M,L) \geq 0$, $\forall (M,L) \in \Omega$. Thus, this proves that disease free equilibrium point E_0 is globally asymptotically stable when $R_0 \leq 1$.

3.6. Disease Endemic Equilibrium Point of the Model. The endemic equilibrium point, E_1, of the model is the steady state solution where leaf curl virus persist in the population of cotton plants. We can obtain by equating each system of the equation equal to zero; that is,

$$\begin{aligned} b_1 - \theta_1 A^* Y^* - \mu A^* &= 0, \\ \theta_1 A^* Y^* - (\mu + \omega) B^* &= 0, \\ b_2 - \theta_2 B^* X^* - \delta X^* &= 0, \\ \theta_2 B^* X^* - \delta Y^* &= 0, \end{aligned} \tag{42}$$

From first equation of (42), we get

$$A^* = \frac{b_1}{\theta_1 Y^* + \mu}. \tag{43}$$

From second equation of (42), we have

$$B^* = \frac{\theta_1 A^* Y^*}{(\mu + \omega)}. \tag{44}$$

Substituting the value of A from equation (43) in to equation (44), we obtain

$$B^* = \frac{b_1 \theta_1 Y^*}{(\mu + \omega)(\theta_1 Y^* + \mu)}. \tag{45}$$

From third equation of (42), we have

$$X^* = \frac{b_2}{\theta_2 B^* + \delta}. \tag{46}$$

Substituting the value of B from equation (45) in to equation (46), we obtain

$$X^* = \frac{b_2(\mu + \omega)(\theta_1 Y^* + \mu)}{b_1 \theta_1 \theta_2 Y^* + \delta(\mu + \omega)(\theta_1 Y^* + \mu)}. \tag{47}$$

From last equation (42), we have

$$\theta_2 B^* X^* = \delta Y^*. \tag{48}$$

Substituting the value B^* from equation (45) and the value X^* from equation (47) in equation (48), we have

$$\frac{b_1 b_2 \theta_1 \theta_2}{b_1 \theta_1 \theta_2 Y^* + \delta(\mu+\omega)(\theta_1 Y^* + \mu)} = \delta. \tag{49}$$

By re arranging and simplifyingbequation (49), we get

$$Y^* = \frac{\mu\delta(\mu+\omega)(R_0^2-1)}{\theta_1(b_1\theta_2 + \delta(\mu+\omega))}. \tag{50}$$

Thus, by substituting equation (50) into equations (43), (45) and (47), we obtain

$$A^* = \frac{b_1(b_1\theta_2 + \delta(\mu+\omega))}{\mu\delta(\mu+\omega)(R_0^2-1) + \mu(b_1\theta_2 + \delta(\mu+\omega))}, \tag{51}$$

$$B^* = \frac{b_1\delta(R_0^2-1)}{\delta(\mu+\omega)(R_0^2-1) + (b_1\theta_2 + \delta(\mu+\omega))}, \tag{52}$$

$$X^* = \frac{b_2(\delta(\mu+\omega)(R_0^2-1) + b_1\theta_2 + \delta(\mu+\omega))}{\delta((b_1\theta_2 + \delta(\mu+\omega))(R_0^2-1) + b_1\theta_2 + \delta(\mu+\omega))} \tag{53}$$

3.7. Local Stability of the Endemic Equilibrium Point. We used the Jacobian stability approach to prove the local stability of the disease endemic equilibrium state in this section.

Theorem 4: When $R_0 > 1$, the model's endemic equilibrium point, E_1, is locally asymptotically stable.

Proof: The local stability of the endemic equilibrium, E_1, is determined based on the signs of the eigenvalues of the Jacobian matrix which is computed at the disease endemic equilibrium, E_1. Now, using the method proposed by [30], the Jacobian matrix of the our model at E_1 is given by:

$$J = \begin{pmatrix} -(\theta_1 Y^* + \mu) & 0 & 0 & -\theta_1 A^* \\ \theta_1 Y^* & -(\mu+\omega) & 0 & \theta_1 A^* \\ 0 & -\theta_2 X^* & -(\theta_2 B^* + \delta) & 0 \\ 0 & \theta_2 X^* & \theta_2 B^* & -\delta \end{pmatrix}, \tag{54}$$

The characteristic equation of Jacobian matrix of equation (54) at disease endemic equilibrium point, E_1 is $|J(E_1) - \lambda I_4| = 0$. That is

$$\begin{vmatrix} -(\theta_1 Y^* + \mu) - \lambda & 0 & 0 & -\theta_1 A^* \\ \theta_1 Y^* & -(\mu+\omega) - \lambda & 0 & \theta_1 A^* \\ 0 & -\theta_2 X^* & -(\theta_2 B^* + \delta) - \lambda & 0 \\ 0 & \theta_2 X^* & \theta_2 B^* & -\delta - \lambda \end{vmatrix} = 0 \tag{55}$$

Equation (55) can be simplified as:

$$P(\lambda) = f_4\lambda^4 + f_3\lambda^3 + f_2\lambda^2 + f_1\lambda + f_0, \tag{56}$$

where

$$\begin{aligned} f_4 =& 1, f_3 = 2\mu + 2\delta + \omega + \theta_1 Y^* + \theta_2 B^*, \\ f_2 =& \mu\delta + (\mu+\delta)(\mu+\omega+\delta)(\mu+\omega)\delta + \\ & (2\delta + \mu + \omega)\theta_1 Y^* + (2\mu + \omega + \delta)\theta_2 B^* + \\ & \theta_1\theta_2(B^* - A^*X^*), \\ f_1 =& \mu\delta(\mu+\omega+\delta) + (\mu+\delta)(\mu+\omega)\delta + \\ & (\mu\delta + (\mu+\delta)(\mu+\omega))\theta_2 B^* + (2\mu + 2\omega + \delta))\delta\theta_1)Y^* \\ & + (\mu+\omega+\delta)\theta_1\theta_2 B^* Y^* - \theta_1\theta_2(\mu+\delta)A^*X^*, \\ f_0 =& \delta\theta_1\theta_2(\mu+\omega)B^*Y^* + \theta_1\delta^2(\mu+\omega)Y^* \\ & + \delta\mu\theta_2(\mu+\omega)B^* + \mu\delta^2(\mu+\omega) - \mu\delta\theta_1\theta_2 A^*X^*. \end{aligned} \tag{57}$$

Now, the characteristic polynomial of equation (56) can be analyzed by Routh-Hurwitz criteria. The coefficients f_4, f_3, f_2, f_1, f_0 of the characteristic polynomial are real positive. Thus, the necessary condition for stability of the disease endemic equilibrium point is fulfilled. Then, the sufficient condition for stability of the system using the Hurwitz array for the characteristic polynomial is presented as follows:

where f_4, f_3, f_2, f_1, f_0 are the coefficients of the characteristic polynomial and the remaining

$$\begin{array}{c|ccc} s^4 & f_4 & f_2 & f_0 \\ s^3 & f_3 & f_1 & 0 \\ s^2 & g_1 & g_2 & g_3 \\ s^1 & h_1 & h_2 & h_3 \\ s^0 & k_1 & k_2 & k_3 \end{array}$$

elements in the array are obtained as follows:

$$g_1 = -\frac{1}{f_3}\begin{vmatrix} f_4 & f_2 \\ f_3 & f_1 \end{vmatrix} = \frac{f_3 f_2 - f_1}{f_3} > 0, g_2 = -\frac{1}{f_3}\begin{vmatrix} f_4 & f_0 \\ f_3 & 0 \end{vmatrix} = f_0, g_3 = -\frac{1}{f_3}\begin{vmatrix} f_4 & 0 \\ f_3 & 0 \end{vmatrix} = 0,$$

$$h_1 = -\frac{1}{g_1}\begin{vmatrix} f_3 & f_1 \\ g_1 & g_2 \end{vmatrix} = \frac{f_1 g_1 - f_3 f_0}{g_1} > 0, h_2 = -\frac{1}{g_1}\begin{vmatrix} f_3 & 0 \\ g_1 & 0 \end{vmatrix} = 0, h_3 = -\frac{1}{g_1}\begin{vmatrix} f_3 & 0 \\ g_1 & 0 \end{vmatrix} = 0,$$

$$k_1 = -\frac{1}{h_1}\begin{vmatrix} g_1 & g_2 \\ h_1 & 0 \end{vmatrix} = f_0, k_2 = -\frac{1}{h_1}\begin{vmatrix} g_1 & 0 \\ h_1 & 0 \end{vmatrix} = 0, k_3 = -\frac{1}{h_1}\begin{vmatrix} g_1 & 0 \\ h_1 & 0 \end{vmatrix} = 0.$$

Since, the coefficients of the characteristic polynomial; f_4, f_3, f_2, f_1, f_0 are real positive and the first column of the Routh-Hurwitz array has the same positive sign. Therefore, by the Routh-Hurwitz criteria all eigenvalues of the characteristics polynomial are negative. Thus, the disease endemic equilibrium point E_1 is locally asymptotically stable if $R_0 > 1$.

3.8. Global Stability of Disease Endemic Equilibrium Point. In this subsection, we will prove the global stability disease endemic equilibrium point.

Theorem 5: For $R_0 > 1$, then the system of equation of (1) at E_1 is globally asymptotical stable.

Proof: To investigate the global stability of the endemic equilibrium point E_1 , we consider the following Lyapunov function for model of equation (1):

$$\begin{aligned} V(t) =& K_1 \frac{(A - A^*)^2}{2} + K_2 \frac{(B - B^*)^2}{2} \\ &+ K_3 \frac{(X - X^*)^2}{2} + K_4 \frac{(Y - Y^*)^2}{2} \end{aligned} \tag{58}$$

where K_1, K_2, K_3, K_4 are chosen. By differentiating (58) with respect to time , we have

$$\begin{aligned}\frac{dV}{dt} =& K_1 (A - A^*) \frac{dA}{dt} + K_2 (B - B^*) \frac{dB}{dt} + \\ & K_3 (X - X^*) \frac{dX}{dt} + K_4 (Y - Y^*) \frac{dY}{dt}\end{aligned} \tag{59}$$

Using system equation (1), equation (59) can becomes,

$$\begin{aligned}\frac{dV}{dt} =& K_1 (A - A^*) [b_1 - (\theta_1 Y + \mu) A] + K_2 (B - B^*) \\ & [\theta_1 AY - (\mu + \omega) B] + K_3 (X - X^*) \\ & [b_2 - (\theta_2 B + \delta) X] + K_4 (Y - Y^*) [\theta_2 BX - \delta Y].\end{aligned} \tag{60}$$

By re arranging equation (60), we have

$$\begin{aligned}\frac{dV}{dt} =& - K_1 (A - A^*) A \left[-\frac{b_1}{A} + (\theta_1 Y + \mu) \right] \\ & - K_2 (B - B^*) B \left[-\frac{\theta_1 AY}{B} + (\mu + \omega) \right] \\ & - K_3 (X - X^*) X \left[-\frac{b_2}{X} + (\theta_2 B + \delta) \right] \\ & - K_4 (Y - Y^*) Y \left[-\frac{\theta_2 BX}{Y} + \delta \right].\end{aligned} \tag{61}$$

Here, we can choose

$$\begin{aligned}K_1 =& \frac{A}{(\theta_1 Y + \mu) A - b_1}, \\ K_2 =& \frac{B}{(\mu + \omega) B - \theta_1 AY}, \\ K_3 =& \frac{X}{(\theta_2 B + \delta) X - b_2} \\ K_4 =& \frac{Y}{\delta Y - \theta_2 BX}.\end{aligned} \tag{62}$$

Hence, we observe that $\frac{dV}{dt} < 0$ and an endemic equilibrium point, E_1, of the model is globally stable. Moreover, $\frac{dV}{dt} = 0$ if and only if either $A = A^*, B = B^*, X = X^*, Y = Y^*$ or $A = B = X = Y = 0$. Thus, using [31], E_1 is global asymptotical stable whenever $R_0 > 1$.

3.9. Sensitivity Analysis of Model Parameters. Sensitivity indices allow us to quantify how much a variable varies when a parameter is changed Rodrigues et al. [32]. The sensitivity index can also be constructed using partial derivatives when the variable is a differentiable function of the parameter.

Definition: The normalized forward sensitivity index of a R_0, that depends differentiably on a parameter, q_i, is given as:

$$\begin{aligned} M_{b_1}^{R_0} &= \frac{\partial R_0}{\partial b_1}.\frac{b_1}{R_0} \\ &= \left(\frac{1}{2\sqrt{\frac{b_1 b_2 \theta_1 \theta_2}{\mu\delta^2(\mu+\omega)}}}\frac{b_2\theta_1\theta_2}{\mu\delta^2(\mu+\omega)}\right).\frac{b_1}{\sqrt{\frac{b_1 b_2 \theta_1 \theta_2}{\mu\delta^2(\mu+\omega)}}} \\ &= \frac{1}{2}. \end{aligned}$$

We determine the sensitivity index of the other parameters using the same technique. table 2 lists the criteria in order of most to least sensitive.

3.10. Interpretation of Sensitivity Indices. Table 2 displays the basic reproductive number sensitivity indices in relation to the important parameters. Positive indices $(b_1, b_1, \theta_1, \theta_2)$ indicate that parameters with increasing values have a significant impact on the spread of the disease. Because the basic reproduction number increases as their values increase, so does the average number of secondary cases of infection. Additionally, those parameters with negative sensitivity indices (μ, δ, ω) have the effect of reducing disease burden when their values rise while the others remain constant. Furthermore, as their values rise, the basic reproduction number decreases, resulting in the disease's endemic areas being reduced.

4. NUMERICAL SIMULATION

We used MATLAB ode45 solvers to numerically validate our work in this section. Our simulations investigate the effect of various model parameter combinations on the transmission dynamics of cotton leaf curl virus (CLCuV). The simulation is run with a wide range of parameter values. The source of the set of parameter values are mainly using assumption. The relevant initial circumstances are used in the simulations and analyses: $A(0) = 800, B(0) = 100, X(0) = 200, Y(0) = 80$ and the parameters values are displayed in Table 2.

TABLE 2. The parameter values and sensitivity index of model.

Parameter	Value	Sensitivity index
b_1	0.97	+ve
b_2	0.27	+ve
θ_1	0.000023	+ve
θ_2	0.00021	+ve
μ	0.0005	-ve
δ	0.0029	-ve
ω	0.019	-ve

The time series plot of state variables for $R_0 < 1$ and $R_0 > 1$ is shown in figure 2 and figure 3. From figure 2, we observe that susceptible cotton individuals are increases asymptotically to the disease free equilibrium point, while the infected cotton individuals are decreases asymptotically to the disease free equilibrium point. Furthermore, the susceptible vector individuals are increases asymptotically to the disease free equilibrium point, while the infected vector individuals are decreases asymptotically to the disease free equilibrium point. In this case, the disease may remove out in the long run. The existence of such condition is due to the fact that $R_0 = 0.2012$ which is less than one. This supports theorem that the stability of disease free equilibrium point exists when $R_0 < 1$, that is if $R_0 < 1$, then on average, one infected cotton plant produces less than one newly infectious plant over the course of its disease period.

From figure 3, we observe that susceptible cotton and vector individuals are decreased due to influence of infected cotton and vector individuals, then they are joins into infected class as a result the infected cotton and vector individuals are increased. Therefore, infected cotton and vectors are increased and the disease endemic equilibrium point exists and stable. The existence of this condition is due to the fact that $R_0 = 3.9277$ which is greater than one. This supports theorem that the stability of disease endemic equilibrium point exists when $R_0 > 1$, that is if $R_0 > 1$, each infected cotton and vectors produces, on average more than one new infected cotton and vectors, then disease will be able to spread in the given area.

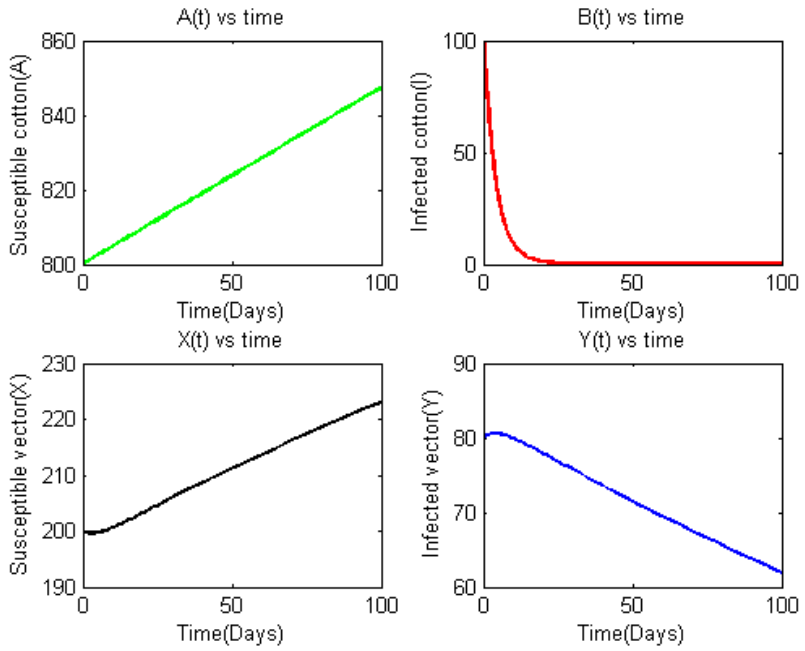

FIGURE 2. Time series plot of state variables for $R_0 = 0.2012 < 1$

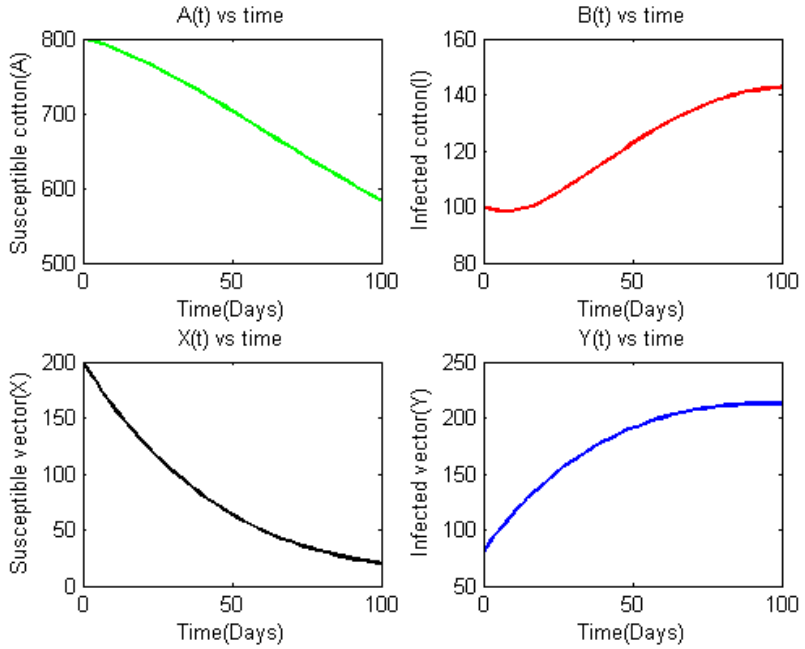

FIGURE 3. Time series plot of state variables for $R_0 = 3.9277 > 1$

Susceptible cotton population (A) and infected cotton population (B) with respect to time t for different values of θ_1 are shown in figure 4. From figure 4, we observe that as infection rate of cotton,θ_1, increases susceptible cotton population (A) decreases while infected cotton population (B) increased. Also, Susceptible vector population (X) and infected vector population (Y) with respect to time t for different values of θ_2 are shown in figure 5. From figure 5, we observe that as the value of infection rate of vector,θ_2, increases susceptible vector population (X) decreases while infected vector population (Y) increased.

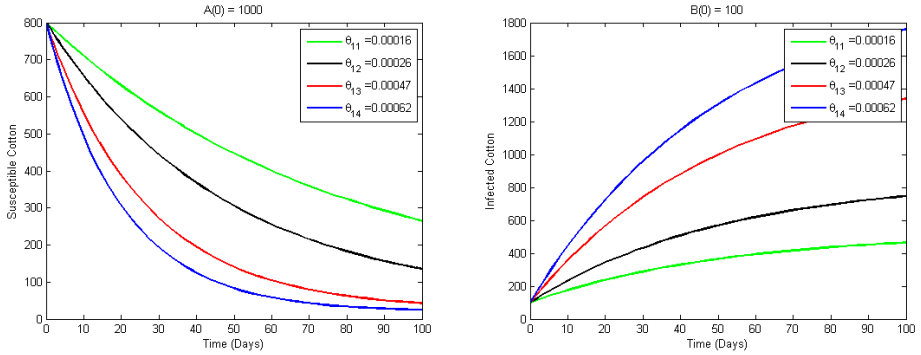

FIGURE 4. Susceptible cotton population (A) and infected cotton population (B) w.r.t. time t for different values of θ_1.

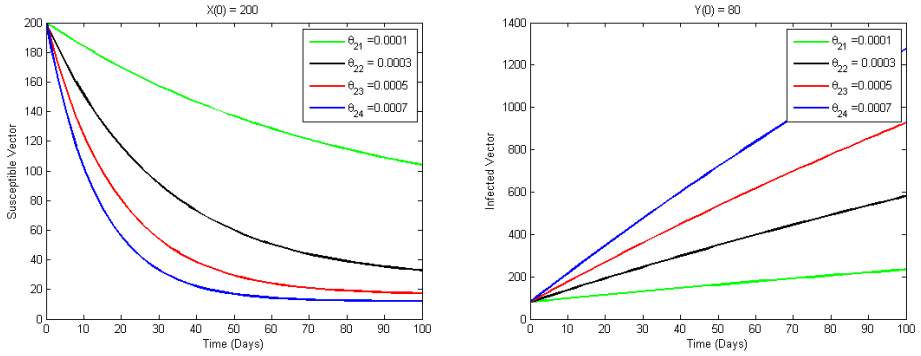

FIGURE 5. Susceptible vector population (X) and infected vector population (Y) w.r.t. time t for different values of θ_2.

5. CONCLUSION

This study established an ecoepidemiological model for the dynamics of Cotton leaf curl virus (CLCuV) illness in cotton. The model's well-posedness, positivity, and boundedness are

all explored. The basic reproduction number was explored, as well as the stability study of the model's cotton equilibria. The cotton-free equilibrium is locally and worldwide asymptotically stable if the basic reproduction number is less than one, but the endemic equilibrium is locally and globally asymptotically stable if the basic reproduction number is more than one. The numerical simulation shows that as infection rate of cotton, θ_1, increases susceptible cotton population (A) decreases while infected cotton population (B) increased. Furthermore, as the value of infection rate of vector, θ_2, increases susceptible vector population (X) decreases while infected vector population (Y) increased.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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