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# Modelling the effects of *tuta-absoluta* on tomato plants

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**Abstract**

A deterministic model is proposed and analyzed to study the dynamics of the effects of *Tuta Absoluta* on tomato plants. Both qualitative and numerical analyses of the model were done and the impact of fumigation on the various epidemiological classes was investigated. Qualitative analysis shows that the model has two equilibria: the disease free equilibrium (DFE) which is locally asymptotically stable when the effective reproduction number is less than one, that is, $R_{es} < 1$ and the endemic equilibrium which is locally asymptotically stable when the effective reproduction number is greater than one, that is, $R_{es} > 1$. Numerical results suggest that when the rate of fumigation is increased, the yield is maintained both in the susceptible and fumigated populations while plants that misses fumigation experiences a drastic yield loss.

*Keywords*: *Tuta Absoluta*; fumigation; equilibria; stability

## 1 Introduction

Tuta-Absoluta (T. Absoluta) is a species of moth in family Gelenchiidae famously known by the common names "tomato leaf miner" and "South American tomato moth". It is capable of producing a devastating yield loss of 100% whether in open fields or protected cultivation [8]. Tuta Absoluta originated from South America and has spread to Europe, Africa and Asia. Since it's detection in Spain in 2006, the pest is continuing to expand its geographical range, including the Sub-Saharan countries. Tuta Absoluta has a high reproductive potential, capable of yielding up to 12 generations per year under optimal conditions. The optimal temperature for its development ranges from 21 to 30°C. Low temperature is a limiting factor for its survival but high temperature is suitable for its development and life span. A mature female can lay up to 260 eggs. The life cycle of this pest comprised of four development stages (egg, larva, pupa, adult): all of which are harmful and can attack different parts (leaves, stems and fruits) of the host plant. The Larva feeds voraciously on tomato plants, producing large galleries in leaves, burrowing in stalks and consuming apical buds and green and ripe fruits. Once introduced, T. Absoluta can be spread with seedlings, infested vines with tomato fruits, tomato fruits and used containers. Outdoor markets vegetable repacking and distribution centers are potential entry or introduction points in the spread of this pest. (CFIA, 2016). Currently, the pest is recognized as a worldwide threat to tomato production [25].

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The spread and advancement of this pest cannot be over-emphasized since its detection in Spain in the year 2006, in the year following it, the pest was detected in France, Italy, Greece, Malta, Morocco, Algeria and Libya. In 2009 it was first reported from Turkey. The epidemic spree of Tuta Absoluta continued to the east to reach Syria, Lebanon, Jordan, Israel, Iraq and Iran. Further advancement of the ravaging pest moved southward reaching Saudi Arabia, Yemen, Oman and the rest of the Persian Gulf states. Africa is no exception, having moved in from the north, Egypt to reach Sudan, South Sudan and Ethiopia from the east and to reach Senegal from the west. It was reported in Nigeria and Zambia in 2016.

Some populations of T. Absoluta have developed resistance to Organophosphate and Pyrethriod pesticides. Newer compounds such as SpinosadImidaclopid and Bacillus thuringiensis have demonstrated some efficacy in controlling European outbreaks of this month. Fortunately, researchers at Cornell University have identified the Sex Pheromone for Tuta Absoluta and has been found to be highly attractive to the male moths. Pheromone lures are used extensively throughout Europe, South America, North America and the Middle East for the monitoring and mass-trapping of the pest. This concept is used to monitor populations of Tuta Absoluta in tomato orchard.

The pheromones are reported to perform well in greenhouses [24]. Although these methods are applied, they are not guaranteed to reduce this pest and may be they are costly and not readily available, especially for small holder tomato farmers in sub-Saharan Africa.
Several chemical pesticides are used to control the pest, but none is suitably adapted for control of the tomato borer due to larvae feeding strategy inside plant tissues and foliar spray easily washed out by wind and rain ([18], [20]). Additionally, most chemical pesticides have adverse impacts to both humans, non-targeted organisms and environment as well [1]. Researchers are now suggesting that pest control using resistant tomato varieties is a good and sustainable option.

Although tomato appears to be the primary host of the Tuta absoluta, it has also been reported to attack other cultivated solanaceous crops such as potato, (Solanumtuberosum L.) and eggplant, (Solanummelongena L.) [8].

The two main pathways of introduction of the Pest is by Larva-infested seedlings from plants and debris including fruits left nearby and through the trade of infested fruits for market. The larva can remain alive in this plant material and re-infect the newly planted crop when the conditions are right. Other wild hosts which are of the family *Solanacea*can also harbour the plant as discussed earlier.

The Moth has a grey-brown colour is approximately 6mm in size and has a wingspan of about 10mm. Newly hatched caterpillars are approximately 0.5mm in size and have a yellowish colour. When maturing, caterpillars turn yellow-green and black band develops behind the head. Fully grown caterpillars are approximately 9mm in size with a pinkish colour on the black. The pupa I s light-brown and approximately 6mm in size.

The most distinctive symptoms of the presence of the species are the blotch-shaped mines (blotch-mines) in the leaves in which the caterpillars can be found. In case of a serious infestation leaves die off completely while mining damage to the stem causing malformation of the plant. Damage to the fruit may give easy access to the disease, causing decay of the fruit.

*In Malta, Tuta Absoluta is now well-established and complete eradication is impossible*. However, it may be controlled by various measures as brought forward earlier, the chief of which is the Pheromone Trap.

Becoming acquainted with disease transmission is of the essence since understanding the dynamics of its spread can help control and put forward a treatment model to that effect. Mathematical Modelling can help put to check through prediction the spread associated with this devastating disease called Tuta Absoluta which can give a yield loss of up to a 100%. It is on this premises that this work is based.

It is pertinent to state that no mathematical model was found in literature for the study of the spread and control of Tuta Absoluta. However, some researchers presented works bordering on the biology of Tuta Absoluta without the design of mathematical models for its study; they include the works in [1], [3], [9], [11], [12], [14], [20] [25].

Consequently, in this paper, a deterministic model is designed and qualitatively analysed. The main objective is to gain insights into the dynamics of the spread of the pest in the presence of fumigation and to explore the scenarios for the effective control of Tuta Absoluta using fumigation.

## 2 Model Formulation

The total population of the plant (tomatoes) at time *t*, denoted by $N(t)$, is split into the mutually exclusive compartments of susceptible plants $(S(t))$, exposed plants to Tuta-Absoluta infection $(E(t))$, Fumigated plant against the infection $(F(t))$, Infected plants with the moth $(I(t))$, Removed plants from the population due to the moth's infection $(R(t))$; the removed class occur, since in literature, plants that are infected with the moths when fumigated can get the moths (burrowing into the plant and thereby destroying it) killed, thus the infected plants are cured of the infection in this way through fumigation. However, it should be mentioned that not many of the plants are cured of the infection once the plants are infected with the moths [20]. Consequently, we account for the few infected plants that are cured after infection due to fumigation with the inclusion of the removed class in the population,

.

$$N(t) = S(t) + F(t) + E(t) + I(t) + R(t).$$

The population of susceptible plants is generated by the introduction of new Tomato seedlings into the nursery or farmland at the rate $\pi$ and diminished by plants which are infested at rate $\beta$. The Population is further reduced by plants who progressed at rate $\alpha$ to the fumigated class and natural death (at a rate $\mu$; natural death occurs in all epidemiological compartments at this rate) so that we have:

$$\frac{dS}{dt} = \pi - \beta IS - \alpha S - \mu S$$

The population of plants in the fumigated class is generated by susceptible plants at the rate of $\alpha$ and this population is diminished by the natural death rate. So that we have:

$$\frac{dF}{dt} = \alpha S - \mu F$$

The population of plants in the exposed class is generated by plants infected by the moth at the rate $\beta$. The population is diminished by the exposed individual who progressed to infected class at rate $\sigma$ and natural death. So that we have:

$$\frac{dE}{dt} = \beta IS - \sigma E - \mu E$$

The population of the infected plants is generated by plants who progressed from the exposed class to the infected class at rate $\sigma$ and it is diminished by plants who die as a result of the disease (disease induced death) at rate $\delta$, it is further reduced by plants who progressed to the removal class at the rate $\varphi$ and natural death.

$$\frac{dI}{dt} = \sigma E - \delta I - \varphi I - \mu I$$

The population of the removal class is generated by plants who progressed from the infected class at rate $\varphi$ and is diminished by natural death.

$$\frac{dR}{dt} = \varphi I - \mu R$$

The flow diagram is as presented below:

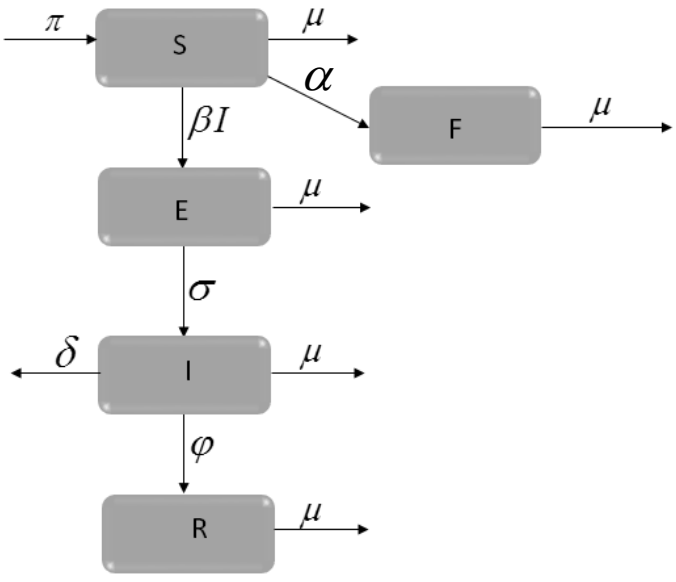



Figure 1 Flow Diagram for the Model

We itemize some of the main assumptions made in the formulation of model (1) as follows:

1. There is no natural recovery from the pest attack at any compartment, this is due to the fact that once a plant is infected with the moths, natural recovery can only take place except when all the ravaging moths die off, regrettably, this cannot occur naturally.
2. There is natural death in all compartments, since some of the plants can die off naturally.
3. We assume that all recruits, that is tomato seedlings and transplanted stalks are not immune to the attack of the pests.
4. There is proximity between the plants since tomatoes just like all other vegetables are planted very close to each other, such that wind blowing can cause the plants to touch each other thereby resulting to interaction of the moths from the infected tomatoes to the other uninfected ones. Through this form of contact, the uninfected plants can get infected by the infected ones.

Based on the above formulations, assumptions and the flow chart, the following deterministic system of non-linear differential equations is the model for the transmission dynamics of Tuta-Absoluta; the associated state variables, parameters and flow diagram of the model are depicted below:

$$\frac{dS}{dt} = \pi - \beta IS - \alpha S - \mu S$$
$$\frac{dF}{dt} = \alpha S - \mu F$$
$$\frac{dE}{dt} = \beta IS - \sigma E - \mu E$$
$$\frac{dI}{dt} = \sigma E - \delta I - \varphi I - \mu I$$
$$\frac{dR}{dt} = \varphi I - \mu R$$

| **Variable** | **Interpretation** |
|---|---|
| S | Susceptible class |
| F | Fumigated class |
| E | Exposed class |
| I | Infected class |
| R | Removed class |
| | |
| **Parameter** | **Interpretation** |
| $\pi$ | Recruitment rate into susceptible class |
| $\beta$ | Contact rate |
| $\alpha$ | Fumigation rate of susceptible plants |
| $\sigma$ | Progression rate to the infected class |

| | |
|---|---|
| $\varphi$ | Progression rate to the removal class |
| $\delta$ | Disease induced death rate |
| $\lambda$ | Force of infection |

Table 1. Description of variables and parameters

Force of infection: The force of infection is given by:

$$\lambda = \beta I .$$

Though we modelled the spread of the disease in plants (tomatoes) and likened it to spread of disease in human population. We are able to do this due to proximity between the plants involved. This proximity between the plants, since tomatoes just like all other vegetables are planted very close to each other, such that wind blowing can cause the plants to touch each other thereby resulting to interaction of the moths from the infected tomatoes to the other uninfected ones. Through this, contact take place between the infected plants and the uninfected ones, thus, the uninfected plants can get infected by the infected ones.

## 3 Invariance Properties

Since the population under study relates to that of physical quantities that cannot be negative, there is a need to prove that all the state variables of the model are non-negative for all time (t), for the model (1) to be epidemiologically meaningful. In other words, the solutions of the model (1) with positive initial data will remain positive for all $t \geq 0$ we do this as follows.

**Lemma 1.** The biologically feasible region:

$$D = \left\{ (S + F + E + I + R) \in R_+^5 : N \leq \frac{\pi}{\mu} \right\}$$

is positively-invariant and attracts all the solutions in $R_+^5$

**Proof:** By adding the equations in the model system (1), we have:

$$\dot{N} \leq \pi - \mu N$$
$$\dot{N} + \mu N \leq \pi.$$

Finding the integrating factors, $(IF) = e^{\int \mu dt} = e^{\mu t}$

$$e^{\mu t} N + \mu N e^{\mu t} \leq \pi e^{\mu t},$$
$$\frac{d}{dt}(N e^{\mu t}) \leq \pi e^{\mu t},$$

Integrating on both sides we get;

$$N e^{\mu t} \leq \frac{\pi}{\mu} e^{\mu t} + c,$$

where c is a constant of integration. Therefore,

$$N \le \frac{\pi}{\mu} + ce^{-\mu t}$$

Using the initial conditions; when $t = 0, N(0) = N_0$.

$$N_0 - \frac{\pi}{\mu} \le c,$$
$$N \le \frac{\pi}{\mu} + (N_0 - \frac{\pi}{\mu})e^{-\mu t}.$$

Applying Birkhoff and Rota's theorem on differential inequality [2], we obtain $0 \le N \le \frac{\pi}{\mu}$ as $t \to \infty$.

Thus, D is a positively invariant set under the flow described by the model (1) so that no solution path leaves through the boundary of region D. Hence, it is sufficient to consider the dynamics of the model (1) in region D. Thus, in this region, the model can be considered as being epidemiologically and mathematically well posed.

# 4 Local Stability of disease free equilibrium

At steady state (equilibrium point), each of the equations are equal to zero.

We have:

$$\pi - (\beta I + \alpha + \mu)S = 0 \qquad f_1$$
$$\alpha S - \mu F = 0 \qquad f_2$$
$$\beta IS - (\sigma + \mu)E = 0 \qquad f_3$$
$$\sigma E - (\varphi + \delta + \mu) = 0 \qquad f_4$$
$$\varphi I - \mu R = 0 \qquad f_5$$

At steady state where there is no infection (or there is absence of disease) is called disease-free equilibrium. That is, a point where $\left(E^* = 0,\ I^* = 0\right)$

$$\pi - (\alpha + \mu)S = 0 \qquad \text{and} \qquad S^* = \frac{\pi}{\alpha + \mu}$$

Therefore, the disease-free equilibrium (DFE) is given by:

$$\varepsilon^0 = \left(S^*, F^*, E^*, I^*, R^*\right) = \left(\frac{\pi}{\alpha + \mu}, 0, 0, 0, 0\right).$$

## 4.1 Basic Reproduction Number

The basic reproduction number $R_0$ is a measurement of the potential for spreading disease in a population. Mathematically, $R_0$ is a threshold parameter for the stability of a disease-free equilibrium and is related to the peak and final size of an epidemic. It is defined as the expected number of

secondary cases of infection which would occur due to a primary case in a completely susceptible population. If $R_0 < 1$, then a few infected individuals introduced into a completely susceptible population will, on average, fail to replace themselves, and the disease will not spread. On the other hand, when $R_0 > 1$, then the number of infected individuals will increase with each generation and the disease will spread.

However, in many disease transmission models, the peak prevalence of the infected hosts and the final size of the epidemic are increasing of $R_0$, making it a useful measure of spread.

The reproduction number can be computed using the next generation operator method on the model (1) equation. Using the $f$ and $V$ denotations, for the new infection and the remaining transmission terms respectively.

$$f = \begin{bmatrix} 0 & \beta S \\ 0 & 0 \end{bmatrix} \qquad \text{and} \qquad V = \begin{bmatrix} P_1 & 0 \\ -\sigma & P_2 \end{bmatrix}$$

Where $P_1 = (\sigma + \mu)$, $P_2 = (\varphi + \delta + \mu)$ and $P_3 = (\alpha + \mu)$

It follows that the reproduction number is denoted by;

$$R_0 = \rho\left(fV^{-1}\right)$$

Where $\rho$ is the spectral radius or the largest eigenvalue of $fV^{-1}$.

$$V^{-1} = \frac{1}{|V|} \text{ adjoint of } V$$

$$V^{-1} = \begin{bmatrix} \frac{1}{P_1} & 0 \\ \frac{\sigma}{P_1 P_2} & \frac{1}{P_2} \end{bmatrix} \qquad \text{and} \qquad fV^{-1} = \begin{bmatrix} \frac{\beta\pi\sigma}{P_1 P_2 P_3} & \frac{\beta S}{P_2} \\ 0 & 0 \end{bmatrix}$$

The eigenvalues of $fV^{-1}$ are:

$$\lambda_1 = \frac{\beta\pi\sigma}{P_1 P_2 P_3} \qquad \text{and} \qquad \lambda_2 = 0$$

Clearly, $\lambda_1$ is the largest eigenvalue and this is the reproduction number.

By substituting the values of $P_1, P_2$ *and* $P_3$ into $\lambda_1$, we have:

$$R_0 = \frac{\beta\pi\sigma}{(\sigma + \mu)(\varphi + \delta + \mu)(\alpha + \mu)}$$

## 4.2 Interpretation of the reproduction number for the model (1)

There is a need to interpret our reproduction number so that we can clearly see meaning of the quantity:

$$R_0 = \frac{\beta\pi\sigma}{(\sigma+\mu)(\varphi+\delta+\mu)(\alpha+\mu)}$$

$$R_0 = \frac{\beta\pi}{(\alpha+\mu)} \times \frac{\sigma}{(\sigma+\mu)} \times \frac{1}{(\varphi+\sigma+\mu)}$$

Where $\frac{\beta\pi}{(\alpha+\mu)}$ is the transmission probability, $\frac{\sigma}{(\sigma+\mu)}$ is that an individual has become infectious (that is moved from the exposed class to the infected class) and $\frac{1}{(\varphi+\sigma+\mu)}$ is the average time that the person is infectious.

**Theorem 2:** The DFE of the model (1) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

**Proof:** To prove this we will consider the Jacobian matrix using equation $f_1, f_2, f_3, f_4$ *and* $f_5$

$$J(S^*, F^*, E^*, I^*, R^*) = \begin{Bmatrix} \frac{\delta f_1}{\delta S} & \frac{\delta f_1}{\delta F} & \frac{\delta f_1}{\delta E} & \frac{\delta f_1}{\delta I} & \frac{\delta f_1}{\delta R} \\ \frac{\delta f_2}{\delta S} & \frac{\delta f_2}{\delta F} & \frac{\delta f_2}{\delta E} & \frac{\delta f_2}{\delta I} & \frac{\delta f_2}{\delta R} \\ \frac{\delta f_3}{\delta S} & \frac{\delta f_3}{\delta F} & \frac{\delta f_3}{\delta E} & \frac{\delta f_3}{\delta I} & \frac{\delta f_3}{\delta R} \\ \frac{\delta f_4}{\delta S} & \frac{\delta f_4}{\delta F} & \frac{\delta f_4}{\delta E} & \frac{\delta f_4}{\delta I} & \frac{\delta f_4}{\delta R} \\ \frac{\delta f_5}{\delta S} & \frac{\delta f_5}{\delta F} & \frac{\delta f_5}{\delta E} & \frac{\delta f_5}{\delta I} & \frac{\delta f_5}{\delta R} \end{Bmatrix}$$

So we have:

$$J(S^*, F^*, E^*, I^*, R^*) = \begin{pmatrix} -(\beta I+\alpha+\mu) & 0 & 0 & -\beta S & 0 \\ \alpha & -\mu & 0 & 0 & 0 \\ \beta I & 0 & -P_1 & \beta S & 0 \\ 0 & 0 & \sigma & -P_2 & 0 \\ 0 & 0 & 0 & \varphi & -\mu \end{pmatrix}$$

Where $P_1 = (\sigma+\mu)$ and $P_2 = (\varphi+\delta+\mu)$

At DFE point $(S^*, F^*, E^*, I^*, R^*) = \left(\frac{\pi}{\mu}, 0, 0, 0, 0\right)$

$$J\left(\frac{\pi}{\alpha+\mu}, 0, 0, 0, 0\right) = \begin{pmatrix} -(\beta I+\alpha+\mu) & 0 & 0 & -\beta S & 0 \\ \alpha & -\mu & 0 & 0 & 0 \\ \beta I & 0 & -P_1 & \beta S & 0 \\ 0 & 0 & \sigma & -P_2 & 0 \\ 0 & 0 & 0 & \varphi & -\mu \end{pmatrix}$$

Reducing the above matrix to a row-echelon form, we have

$$J\left(\frac{\pi}{\alpha+\mu},0,0,0,0\right)=\begin{Bmatrix} -P_3 & 0 & 0 & -\beta S & 0 \\ 0 & -\mu P_3 & 0 & -\beta S\alpha & 0 \\ 0 & 0 & -P_1 & \frac{\beta\pi}{P_3} & 0 \\ 0 & 0 & 0 & \frac{\beta\pi\sigma - P_1P_2P_3}{P_1P_2P_3} & 0 \\ 0 & 0 & 0 & 0 & -\mu \end{Bmatrix}$$

The eigenvalues of the above matrix are

$$\lambda_1=-P_3, \qquad \lambda_2=-\mu P_3<0, \qquad \lambda_3=-P_1<0$$

$$\lambda_4=\frac{\beta\pi\sigma-P_1P_2P_3}{P_1P_2P_3}[R_0-1], \qquad \lambda_5=-\mu$$

Here, all the eigenvalues are clearly negative except $\lambda_4$, but if $R_0<1$ then $\lambda_4$ will be negative. Therefore, the DFE point is locally asymptotically stable if $R_0<1$ and unstable if $R_0>1$.

## 5 Global Stability

In this section we examine the global stability of the model. We consider the following theorem.

**Theorem 3:** The DFE of the model (1) is globally asymptotically stable if $R_0 \le 1$ and unstable if $R_0 \ge 1$

**Proof:** To prove this we will consider following Lyapunov function;

$$\dot{F}=a\dot{E}+\dot{I}b$$

With Lyapunov derivative (where a dot represents differentiation with respect to time)

$$\dot{F}=a\dot{E}+\dot{I}b$$

With a little perturbation from the reproduction number we have; $a=\sigma$ and $b=P_1$

Therefore

$$\dot{F}=a(\beta IS-P_1E)+b(\sigma E-P_2I)$$

$$\dot{F}=\sigma(\beta IS-P_1E)+P_1(\sigma E-P_2I)$$

$$\dot{F}=\beta I\sigma S-P_1\sigma E+P_1\sigma E-P_1P_2I$$

$$\dot{F}=I(\beta\sigma S-P_1P_2)$$

$$\dot{F}=P_1P_2I\left(\frac{\beta\sigma S}{P_1P_2}-1\right) \qquad \forall S=\frac{\pi}{\alpha+\mu}=\frac{\pi}{P_3}\le N$$

$$\dot{F} \le P_1 P_2 I[R_0 - 1] \qquad \forall R_0 \le 0$$

Since all the model parameters are non-negative, it follows that $F \le 0$ for $R_0 \le 1$ with $F = 0$ , if and only if $E = I = 0$. Hence, $F$ is a Lyapunov function in the invariant region. Therefore, by the LaSalle's invariance principle, every solution to the equation in the model, with condition in the invariant region, approaches $\mathcal{E}_0$ as $t \to \infty$. Thus, the above theorem shows that the classical epidemiological requirement of $R_0 \le 1$ is the necessary and sufficient condition for the elimination of the disease from the community.

## 5.1 Endemic Equilibrium (EE)

The endemic equilibrium can be obtained when $(S, F, E, I, R) \neq 0$. To obtain the conditions necessary for the existence of endemic equilibrium point, the model (1) is solved in terms of the force of infection $\lambda = \beta I$, we do this as follows:

$$S^* = \frac{\pi}{(\alpha + \mu)}, \qquad E^* = \frac{\beta I S}{(\sigma + \mu)} \quad \text{and} \quad I^* = \frac{\sigma E}{(\varphi + \delta + \mu)}$$

From $\lambda = \beta I$ (1)

$$E^* = \frac{\beta I \pi}{(\sigma + \mu)(\varphi + \delta + \mu)} = \beta \pi \left[ \frac{\sigma E^*}{(\varphi + \delta + \mu)} \right] \times \left[ \frac{1}{(\sigma + \mu)\left( \frac{\sigma E^*}{(\varphi + \delta + \mu)} + \frac{(\alpha + \mu)}{1} \right)} \right]$$

$$E^* = \frac{\beta \pi \sigma E^*}{(\varphi + \delta + \mu)} \times \frac{(\varphi + \delta + \mu)}{(\sigma + \mu)\left( \beta \sigma E^* + (\alpha + \mu)(\varphi + \sigma + \mu) \right)}$$

$$1 = \frac{\beta \pi \sigma}{(\sigma + \mu)\left( \beta \sigma E^* + (\alpha + \mu)(\varphi + \sigma + \mu) \right)}$$

$$\beta \pi \sigma = (\sigma + \mu)\left( \beta \sigma E^* \right) + (\sigma + \mu)(\alpha + \mu)(\varphi + \sigma + \mu)$$

$$\beta \sigma E^* = \frac{\beta \pi \sigma - (\sigma + \mu)(\alpha + \mu)(\varphi + \sigma + \mu)}{(\sigma + \mu)}$$

Let $P_1 = (\sigma + \mu)$, $P_2 = (\varphi + \delta + \mu)$ and $P_3 = (\alpha + \mu)$

$$E^* = \frac{\beta \pi \sigma - P_1 P_2 P_3}{\beta \sigma P_1} \qquad (2)$$

$$I^* = \frac{\sigma}{P_2}\left( \frac{\beta \pi \sigma - P_1 P_2 P_3}{\beta \sigma P_1} \right)$$

$$I^* = \frac{\beta\pi\sigma - P_1P_2P_3}{\beta P_1P_2} \tag{3}$$

Substituting (3) into force of infection $\lambda = \beta I$, we get;

$$\lambda^* = \beta\left(\frac{\beta\pi\sigma - P_1P_2P_3}{\beta P_1P_2}\right) \quad \text{and} \quad \lambda^* = \left(\frac{\beta\pi\sigma - P_1P_2P_3}{P_1P_2}\right)$$

$$\lambda^* = \left(\frac{\beta\pi\sigma}{P_1P_2} - P_3\right) = P_3\left[\frac{\beta\pi\sigma}{P_1P_2P_3} - 1\right]$$

$$\lambda^* = P_3\left(R_0 - 1\right) \tag{4}$$

Clearly, from (4) for $\lambda^*$ to have a unique value, $R_0$ must be greater than one, that is $R_0 > 1$.

## 6 Numerical Simulation

The parameters in table 1 will be used in the Simulation. The objective of these simulations is to illustrate some of the theoretical results obtained in this study.

| S/N | Parameters | Value | Source |
|---|---|---|---|
| 1 | π | 15 per year | Estimated |
| 2 | β | 0.35 per year | Estimated |
| 3 | σ | 0.27 per year | Estimated |
| 4 | δ | 0.30 per year | Estimated |
| 5 | $\alpha_1$ | 0.32 per year | Estimated |

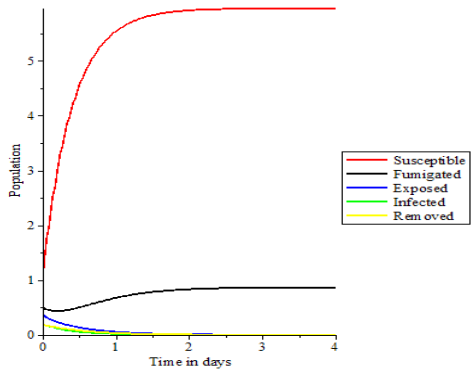
| | | | |
|---|---|---|---|
| 6 | $\alpha_2$ | 2.32 per year | Estimated |
| 7 | μ | 2.2 per year | Estimated |
| 8 | φ | 0.56 per year | Estimated |

Table 2. Parameter values

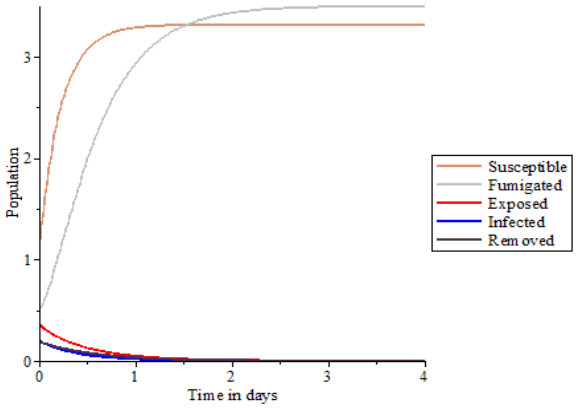
Under this section we will discuss the effect of fumigation rate (α) on the dynamics of Tuta Absoluta. This being that Tuta Absoluta thrives, spreads and inevitably devastates with or without pesticides. It however affects the yield loss.

1. Effect of fumigation rate ($\alpha_1$) on the Model (General case (a)), fig 2a
2. Effect of fumigation rate ($\alpha_2$) on the Model (General case (b)) fig 2b

(a)




(b)

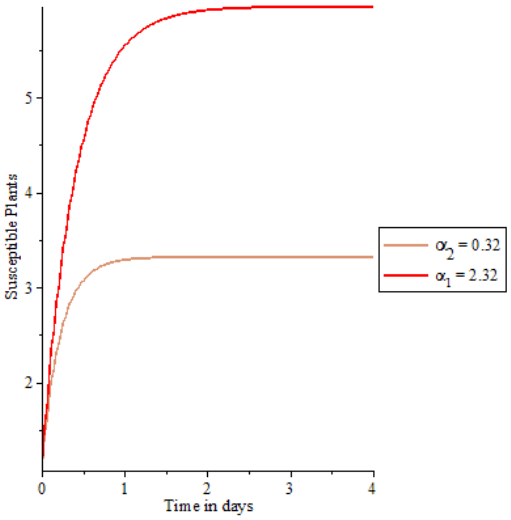


It is noticed that fumigation has a major role to play in curbing the spread of Tuta Absoluta from reaching maximum potential.

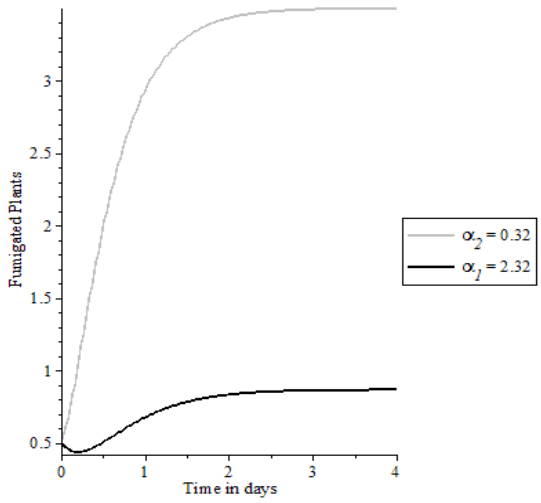
We observe that in other to control Tuta Absoluta, Fumigation must be emphasized because the rate of fumigation affects the population of plants in the classes: exposed, fumigated, infected, and removed.

In this section, we are varying the fumigation rate (α) in each class as below:

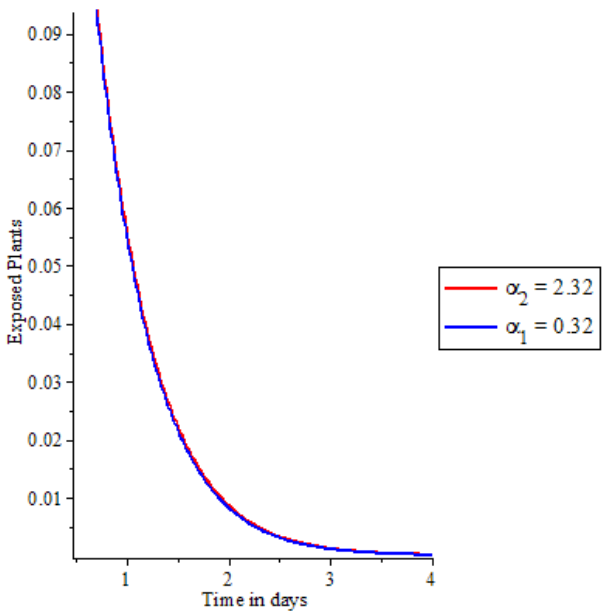
(c)



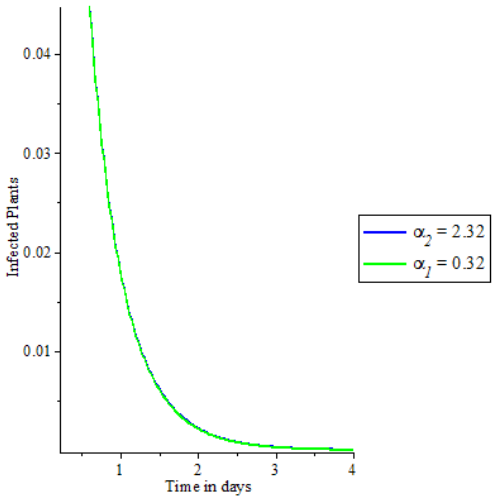

(d)




(e)



(f)




(g)

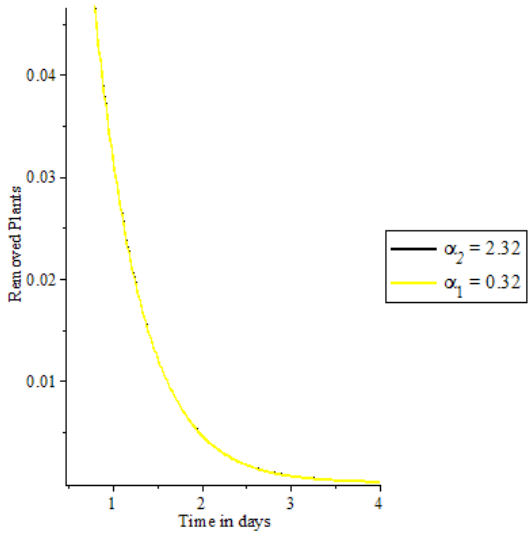



Figure 2

The plots in Figure 2 showing the change in the dynamics of each sub-population with respect to time by varying the fumigation rate of the Tuta Absoluta model where (c) susceptible population, (d) fumigated population, (e) exposed population, (f) infected population, (g) removed population.

The above show us the effect of Fumigation on each of these classes; invariably it affects the population of tomatoes.

In Figure 2(c), it is observed that the increase in the rate of fumigation on the susceptible population, the slower the rate of progression to complete yield loss. The lower the rate of fumigation, the faster the progression to complete yield loss. In Figure 2(d), it is observed that among the fumigated plants and non-fumigated ones, the difference is clear, in the sense that, among plants with increased fumigation,z the loss is invariably lower to those with a lower rate of fumigation.

Figure 2(e): It is observed that among the exposed plants, it is seen that as many plants progress from the susceptible class to the exposed class, at increased fumigation rate, it decreases the time it takes to absolute or total yield loss.

Figure 2(f): It is observed that, when the level of fumigation is increased among the infected individuals, it is practically the same as the exposed individuals because according to the biology of the disease, plants exposed to Tuta Absoluta are as good as dead. Fumigation actually plays little or no role in this class.

Figure 2(g): At high fumigation, as earlier mentioned in the exposed and infected class, plants at this stage are practicably irrecoverable.

However, in the susceptible and fumigated class we note that plants are shielded from T. Absoluta, whereas in the exposed, infected and removed class there are brought to zero yields in a matter of days.

## Conclusion

Based on the discussions above, it is observed and hereby concluded that as long as there are no interventions (fumigation) to control the spread of the disease, the disease will not be wiped out of the population. We finally observe that when fumigation is applied to the plants at early stages the yield loss is drastically reduced and Tuta Absoluta will not be as devastating as it usually is.

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