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Mathematical model to assess the impacts of aflatoxin contamination in crops, livestock and humans

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ABSTRACT

Aflatoxin contamination poses a significant challenge in food safety and security as it affects both the health of consumers and supply chains. Due to the health impacts associated with aflatoxin contamination, countries have set standards and restrictions for importing food crops and animal feed, resulting in greater economic losses to farmers, transporters, and crop processors. This study aimed to develop a mathematical model that tracks the contamination status of crops, livestock and humans in supporting efforts to control aflatoxin. The analysis of the mathematical model shows that both aflatoxin contamination-free equilibrium (ACFE) and aflatoxin contamination-persistence equilibrium (ACPE) exist. To study the dynamics of contamination, we derived the basic aflatoxin contamination number, R_0 which is analogous to the basic reproduction number in epidemiological models. When $R_0 < 1$, the ACFE is globally asymptotically stable, whereas when $R_0 > 1$ the ACPE is globally asymptotically stable. Partial Rank Correlation Coefficients (PRCCs) for global sensitivity analysis were calculated using Latin Hypercube Sampling (LHS) to see how sensitive and significant the parameter is on each variable. Results from numerical simulations showed that decreasing crop contamination and shading rates and increasing the death rate of aflatoxin fungi in soil by 50% can reduce the basic contamination number by above 92%. Thus, it is important to introduce control measures that target crop contamination, shading and death rates of aflatoxin fungi in soil to reduce contamination in the population. Compared to other studies in aflatoxin contamination, the current study provides a thoroughly global sensitivity analysis of parameters involved in contamination and indicated the most important ones for control strategies.

Introduction

Aflatoxins are poisonous substances that are produced by *Aspergillus flavus* and *Aspergillus parasiticus* (certain types of fungi) that are found naturally all over the world and can grow in soil, decaying vegetation, and grains if conditions are favorable [1,2]. Temperatures around 30 °C, relative humidity between 80% and 85%, and other factors such as water activity and soil pH are ideal for Aspergillus to grow and produce aflatoxin [2].

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There are more than 20 different types of aflatoxins, but the most important are B_1 , B_2 , G_1 and G_2 with B_1 being the most prevalent in food crops and having greater toxicity [2,3]. When contaminated food is processed, aflatoxins enter the general food supply, where they can be found in both human food and feed for agricultural animals. Livestock fed on contaminated food can also pass aflatoxin onto eggs, milk products and meat [4]. Humans become contaminated in different ways: first, by consuming contaminated food crops like maize, peanuts, rice and other related crops, or by consuming products made from contaminated crops. Second, through consuming products of contaminated livestock like eggs, meat and dairy products [5]. Other ways of contamination include: inhaling dust generated during handling or processing of contaminated crops or feeds [6,7] and mother to child through breastfeeding [5], although these are negligible ways of contamination and have not been considered in this study [6,7].

Aflatoxin contamination has been reported to have effects on the health of consumers and business chains. Consuming high doses of aflatoxin in a short period of time can cause acute aflatoxicosis, leading to death [8]. Intake of low to moderate doses of aflatoxins over a prolonged time period results in immunity suppression, children's impaired growth and liver cancer [2,9]. Among these effects, liver cancer is the most severe and well known [8]. Estimates from the literature show that among 782,200 new cases of liver cancer per year globally, 648,200 (83%) occur in developing countries [10]. According to [8,11] more than 28.2% of the annual global liver cancer cases are associated with aflatoxin contamination, and 40% occur in Africa.

Health impacts associated with aflatoxin contamination have forced countries to set standards and restrictions for importing food crops and animal feed, resulting in greater economic losses for farmers, transporters, and crop processors. For example, the European Union allows maize and groundnuts with aflatoxin concentrations below $5\mu g/kg$ and $8\mu g/kg$, respectively, to be imported and consumed as food or feed [12]. The East African Community allows maize with below $10\mu g/kg$ to facilitate an equal standard in importing and exporting maize among member countries [13]. The Food and Agriculture Organization of the United Nations (FAO) estimates that more than 25% of the world's food crops exceed the aflatoxin standards and are destroyed each year [5,6]. It is estimated that aflatoxin contamination causes losses to the corn/maize industry ranging from USD 52.1 million to USD 1.68 billion annually in the United States [14]. In Africa, aflatoxin contamination causes losses of more than USD 750 million annually [15].

Mathematical models can be used to study the dynamics of aflatoxin contamination from food crops to livestock and humans. With these models, different control strategies can be tested and simulated to ascertain their effectiveness before implementation. Models help to identify high and low risk areas based on historical data, the life cycle of aflatoxin-causing fungi, and environmental and weather factors. The types of mathematical models used range from empirical to mechanistic models [16,17]. Empirical models are based on statistical analysis of data observed in field experiments to establish the relationship between yields or aflatoxin contamination and climate variables. On the other hand, mechanistic models are based on cause-and-effect relationships among variables to represent biological, chemical, or physical processes [16,17]. In practice, model development can involve both approaches.

Many statistical models have been developed to predict aflatoxin contamination using weather and environmental data. These include Afla-maize model [16], Baranyi model [18], aflatoxin simulation model [19], logistic regression models [9,20] and Pitt model [21]. However, all statistical models aim at prediction and the relationship between aflatoxin contamination and weather and environmental data. They do not capture the process behind aflatoxin contamination and do not simulate important parameters. It is therefore difficult to simulate control strategies for reducing aflatoxin contamination in crops, livestock and humans. In mathematics, the dynamics of systems are usually analyzed using differential equations. [22,23] used differential equations to explain the dynamics of toxicity associated with aflatoxins and their control using probiotics, respectively. However, they considered plants as one population, animals as one population and humans as one population which limited exploration of incidents in each sub-population.

The aim of the current study is to develop a mathematical model to assess the impact of aflatoxin contamination on crops, livestock and humans. We split the populations and use susceptible and contaminated sub-populations in each population of crops, livestock and humans. We perform global sensitivity analysis of parameters with their corresponding Partial Rank Correlation Coefficients (PRCCs) using Latin Hypercube Sampling (LHS) to show the significance of all parameters for each variable.

Model formulation

Humans, livestock and crops are divided into subgroups or compartments depending on the status of aflatoxins contamination. The human population is divided into susceptible $S_H(t)$ and contaminated $C_H(t)$ subgroups, the livestock population is also divided into susceptible $S_L(t)$ and contaminated $C_L(t)$ subgroups and the crop harvests are divided into susceptible $S_C(t)$ and contaminated $C_C(t)$ subgroups. Another compartment is Aflatoxin fungi A(t) which represent the amount of *Aspergillus flavus* and *Aspergillus parasiticus* per unit volume in soil at time t. Individuals in the susceptible group have not been contaminated by aflatoxin and thus they are aflatoxicosis free. In contaminated groups, individuals have been contaminated with aflatoxin.

Crops are recruited at crops' production rate of π_C . Aflatoxins are produced when susceptible crops contact *Aspergillus flavus* and *Aspergillus parasiticus* fungi provided there are favorable conditions. Susceptible crops are contaminated at a force of contamination function λ_1 which is defined by Eq. (1).

$$\lambda_1 = \beta_1 A,\tag{1}$$

where β_1 is the contamination rate of susceptible crops.

Livestock are recruited at the rate of π_L through birth. Susceptible livestock acquire aflatoxin through consumption of contaminated feeds and become contaminated at a force of contamination function λ_2 defined by Eq. (2).

$$\lambda_2 = \beta_2 C_C,\tag{2}$$

where β_2 is the contamination rate susceptible livestock from contaminated crops.

Susceptible humans acquire aflatoxin directly through consumption of contaminated food and indirectly through consumption of products of contaminated livestock at a force of contamination function λ_3 defined by Eq. (3).

$$\lambda_3 = \beta_4 C_C + \beta_5 C_L,\tag{3}$$

where β_4 is the contamination rate of humans from contaminated crops and β_5 is the contamination rate of humans from contaminated livestock. Other ways of transmission have very minimal effects on the dynamics of contamination and therefore excluded from the current study. In formulating a mathematical model, we assumed the following:

- (i) The soil is a reservoir of aflatoxin-producing fungi and can be maintained for a prolonged period.
- (ii) Contaminated crops add aflatoxin fungi to the soil.
- (iii) Aflatoxin-producing fungi invade crops from the soil and form aflatoxin when there are favorable conditions.
- (iv) Susceptible livestock get aflatoxins only after consuming contaminated crops.
- (v) Susceptible humans get aflatoxins after consuming contaminated crops, livestock, or their contaminated products. Other ways of transmission are very minimal and therefore excluded.
- (vi) The death rates for livestock and humans are not higher than their birth rates.
- (vii) Once crops, livestock and humans are contaminated with aflatoxin, they cannot be decontaminated completely.
- (viii) All recruits in each population are susceptible to aflatoxin contamination.
- (ix) Each population is homogeneously mixed.

Based on the dynamics of the aflatoxin contamination, model assumptions, definition of variables and parameters, the dynamics of aflatoxin contamination is summarized in the flow diagram shown by Fig. 1. Guided by the assumptions and the flow diagram in Fig. 1 the dynamics of aflatoxin contamination is summarized by the system of Eqs. (4).

$$\begin{cases} \frac{dS_C}{dt} &= \pi_C - \beta_1 A S_C - \omega_1 S_C - \mu_C S_C, \\ \frac{dC_C}{dt} &= \beta_1 A S_C - \omega_1 C_C - \mu_C C_C, \\ \frac{dS_L}{dt} &= \pi_L - \beta_2 C_C S_L - \mu_L S_L, \\ \frac{dC_L}{dt} &= \beta_2 C_C S_L - (\mu_L + \phi_L) C_L, \\ \frac{dS_H}{dt} &= \pi_H - \beta_4 C_C S_H - \beta_5 C_L S_H - \mu_H S_H, \\ \frac{dC_H}{dt} &= \beta_4 C_C S_H + \beta_5 C_L S_H - (\mu_H + \phi_H) C_H, \\ \frac{dA}{dt} &= \rho C_C - \alpha A, \end{cases}$$
(4)

subject to the following non-negative initial conditions $S_C > 0$, $C_C \ge 0$, $S_L > 0$, $C_L \ge 0$, $S_H > 0$, $C_H \ge 0$ and $A \ge 0$.

Model analysis

In this section we perform the analysis of the model by considering both positivity of solutions and model boundedness. The equilibrium points and their stability are also discussed in this section.

Positivity of solutions

We demonstrate that the solution of model system (4) remain positive for all non-negative initial conditions in invariant region using the approach by [24].

Taking the first equation of system (4),

$$\frac{dS_C}{dt} = \pi_C - \beta_1 A S_C - \omega_1 S_C - \mu_C S_C.$$
(5)

Let $S_C(0) > 0$.

Now suppose that $\exists t = t_0 > 0$ such that $S_C(t_0) = 0$, $\frac{dS_C(t_0)}{dt} < 0$, $C_C(t_0) \ge 0$, $S_L(t_0) > 0$, $C_L(t_0) \ge 0$, $S_H(t_0) > 0$, $C_H(t_0) \ge 0$ and $A(t_0) \ge 0$.

Thus,

$$\frac{dS_C(t_0)}{dt} = \pi_C - \beta_1 A(t_0) S_C(t_0) - \omega_1 S_C(t_0) - \mu_C S_C(t_0) = \pi_C > 0.$$
(6)

Which is a contradiction. Therefore, $S_C(t) > 0 \forall t$. Taking the second equation of system (4),

$$\frac{dC_C}{dt} = \beta_1 A S_C - \omega_1 C_C - \mu_C C_C. \tag{7}$$



Fig. 1. Aflatoxin contamination dynamics compartmental flow diagram.

Let $C_C(0) \ge 0$.

Now suppose that $\exists t = t_1 > 0$ such that $C_C(t_1) = 0$, $\frac{dC_C(t_1)}{dt} < 0$, $S_C(t_1) > 0$, $S_L(t_1) > 0$, $C_L(t_1) \ge 0$, $S_H(t_1) > 0$, $C_H(t_1) \ge 0$ and $A(t_1) \ge 0$.

$$\frac{dC_C(t_1)}{dt} = \beta_1 A(t_1) S_C(t_1) - \omega_1 C_C(t_1) - \mu_C C_C(t_1) \ge 0$$
(8)

Which is a contradiction. Therefore, $C_C(t) \ge 0 \forall t$.

The positivity of all other variables in system (4) can be proved using the same approach. Therefore, we conclude that: $S_C > 0$, $C_C \ge 0$, $S_L > 0$, $C_L \ge 0$, $S_H > 0$, $C_H \ge 0$ and $A \ge 0$ for model system (4). The positivity of solution has the physical meaning that susceptible crops, livestock and humans cannot be zero since they are recruited each year. However, contaminated crops, livestock, humans and aflatoxin fungi can be zero when there is no contamination in populations.

Boundedness of the system

(10

Model system (4) can be divided into the following independent sub-systems:

$$\begin{cases} \frac{dS_C}{dt} = \pi_C - \beta_1 A S_C - \omega_1 S_C - \mu_C S_C, \\ \frac{dC_C}{dt} = \beta_1 A S_C - \omega_1 C_C - \mu_C S_C, \end{cases}$$
(9)

$$\begin{cases} \frac{dS_L}{dt} = \pi_L - \beta_2 C_C S_L - \mu_L S_L, \end{cases}$$
(10)

$$\begin{cases} \frac{dC_L}{dt} = \beta_2 C_C S_L - (\mu_L + \phi_L) C_L. \\ \begin{cases} \frac{dS_H}{dt} = \pi_H - \beta_4 C_C S_H - \beta_5 C_L S_H - \mu_H S_H, \\ \frac{dC_H}{dt} \end{cases}$$
(11)

$$\frac{d^2 H}{dt} = \beta_4 C_C S_H + \beta_5 C_L S_H - (\mu_H + \phi_H) C_H.$$

From sub-system (9), adding the equations we have $N_C = S_C + C_C$. It can be shown that

$$\frac{dN_C}{dt} = \pi_C - (\omega_1 + \mu_C)N_C - (\omega_1 + \mu_C)C_C \le \pi_C - (\omega_1 + \mu_C)N_C,$$
(12)

Using the theory of differential inequality, we separate variables and solve for $N_C(t)$. We obtain

$$N_{C}(t) \leq \frac{\pi_{C}}{(\omega_{1} + \mu_{C})} - \left(\frac{\pi_{C}}{(\omega_{1} + \mu_{C})} - N_{C}(0)\right) e^{-(\omega_{1} + \mu_{C})t},$$
(13)

F.A. Mgandu et al.

It follows that $\lim_{t\to\infty} \sup(N_C(t)) \le \frac{\pi_C}{\omega_1 + \mu_C}$. Since $N_C(t) = S_C(t) + C_C(t)$, we have that each of the individual state variables is east than or equal to $\frac{\pi_C}{\omega_1 + \mu_C}$.

The second regular to
$$\frac{1}{\omega_1 + \mu_C}$$
.

Same approach is used for sub-systems (10) and (11). Adding equations we have $N_L = S_L + C_L$ and $N_H = S_H + C_H$, respectively. Therefore,

$$\frac{dN_L}{dt} = \pi_L - \mu_L N_L - (\mu_L + \phi_L)C_L \le \pi_L - \mu_L S_L,$$
(14)

$$\frac{dN_H}{dt} = \pi_H - \mu_H N_H - (\mu_H + \phi_H) C_H \le \pi_H - \mu_H S_H.$$
(15)

It follows that
$$\lim_{t\to\infty} sup(N_L(t)) \le \frac{\pi_L}{\mu_L}$$
 and $\lim_{t\to\infty} sup(N_H(t)) \le \frac{\pi_H}{\mu_H}$.

Considering the last equation of model system (4) for aflatoxin fungi in the environment, we have:

$$\frac{dA}{dt} = \rho C_C - \alpha A \tag{16}$$

Since the total amount of crops $S_C + C_C \le \pi_C / (\omega_1 + \mu_C)$, it can be concluded that $C_C \le \pi_C / (\omega_1 + \mu_C)$. Thus,

$$\frac{dA}{dt} \le \frac{\rho \pi_C}{\omega_1 + \mu_C} - \alpha A. \tag{17}$$

Using same approach, we separate variables and solve for A. It follows that,

$$\lim_{t \to \infty} \sup(A(t)) \le \frac{\rho \pi_C}{\alpha(\omega_1 + \mu_C)}$$
(18)

So, it can be concluded that the set

$$\begin{split} \Omega &= \left\{ (S_C, C_C, S_L, C_L, S_H, C_H, A) \in \mathbb{R}^7_+ | 0 \le S_C + C_C \le \frac{\pi_C}{\omega_1 + \mu_C}, 0 \le S_L + C_L \le \frac{\pi_L}{\mu_L}, \\ 0 \le S_H + C_H \le \frac{\pi_H}{\mu_H}, 0 \le A \le \frac{\rho \pi_C}{\alpha(\omega_1 + \mu_C)} \right\} \end{split}$$

is bounded with respect to model system (4). Therefore, Ω is a feasible region for model system (4).

Equilibrium points and stability

In this section, we assess the existence of equilibrium points, contamination number and stability of equilibrium points.

Aflatoxin contamination free equilibrium (ACFE) point

To obtain an aflatoxin contamination-free equilibrium point, the right side of equations in model system (4) is set to zero. All forces of contamination in steady state are set to zero; $C_C = C_L = C_H = A = 0$. Upon computation the aflatoxin contamination-free equilibrium point is denoted by E^0 and is given by:

$$E^{0}(S_{C}^{0}, C_{C}^{0}, S_{L}^{0}, C_{L}^{0}, S_{H}^{0}, C_{H}^{0}, A^{0}) = \left[\frac{\pi_{C}}{\omega_{1} + \mu_{C}}, 0, \frac{\pi_{L}}{\mu_{L}}, 0, \frac{\pi_{H}}{\mu_{H}}, 0, 0\right]$$

Basic aflatoxin contamination number

1

The basic aflatoxin contamination number, R_0 is analogous to the basic reproduction number in epidemiological models, a threshold quantity used to examine equilibrium points. It is a crucial quantity that defines how contamination behaves. If $R_0 > 1$, aflatoxin contamination persists and if $R_0 < 1$, aflatoxin contamination diminishes. The study adopts next generation matrix technique to establish the basic aflatoxin contamination number. Consider system (19) of contaminated variables:

$$\begin{cases} \frac{dC_C}{dt} &= \beta_1 A S_C - \omega_1 C_C - \mu_C C_C, \\ \frac{dC_L}{dt} &= \beta_2 C_C S_L - (\mu_L + \phi_L) C_L, \\ \frac{dC_H}{dt} &= \beta_4 C_C S_H + \beta_5 C_L S_H - (\mu_H + \phi_H) C_H, \\ \frac{dA}{dt} &= \rho C_C - \alpha A. \end{cases}$$
(19)

The basic contamination number is obtained by finding the spectral radius of the next generation matrix:

$$FV^{-1} = \left[\frac{\partial \mathcal{F}_i(E^0)}{\partial t}\right] \left[\frac{\partial \mathcal{V}_i(E^0)}{\partial t}\right]^{-1}$$

where E^0 is an aflatoxin contamination free equilibrium point, the vector \mathcal{F}_i refers to new aflatoxin contamination appearance rate in compartment *i* while vector \mathcal{V}_i is the transfer of contamination out of compartment *i*, such that:

$$\mathcal{F}_{i} = \begin{bmatrix} \beta_{1}AS_{C} \\ \beta_{2}C_{C}S_{L} \\ \beta_{4}C_{C}S_{H} + \beta_{5}C_{L}S_{H} \\ 0 \end{bmatrix}, \quad \mathcal{V}_{i} = \begin{bmatrix} (\omega_{1} + \mu_{C})C_{C} \\ (\mu_{L} + \phi_{L})C_{L} \\ (\mu_{H} + \phi_{H})C_{H} \\ -\rho C_{C} + \alpha A \end{bmatrix}$$

The Jacobian matrix of \mathcal{F}_i and \mathcal{V}_i at E^0 , the variational matrices F and V are obtained respectively.

$$F = \begin{bmatrix} 0 & 0 & 0 & \frac{\beta_1 \pi_C}{\omega_1 + \mu_C} \\ \frac{\pi_L \beta_2}{\mu_L} & 0 & 0 & 0 \\ \frac{\pi_H \beta_4}{\mu_H} & \frac{\pi_H \beta_5}{\mu_H} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix},$$

$$V = \begin{bmatrix} \omega_1 + \mu_C & 0 & 0 & 0 \\ 0 & \phi_L + \mu_L & 0 & 0 \\ 0 & 0 & \phi_H + \mu_H & 0 \\ -\rho & 0 & 0 & \alpha \end{bmatrix}.$$
(20)
(21)

The inverse of variational matrix V becomes;

$$V^{-1} = \begin{bmatrix} \left(\omega_1 + \mu_C\right)^{-1} & 0 & 0 & 0 \\ 0 & \left(\phi_L + \mu_L\right)^{-1} & 0 & 0 \\ 0 & 0 & \left(\phi_H + \mu_H\right)^{-1} & 0 \\ \frac{\rho}{\left(\omega_1 + \mu_C\right)\alpha} & 0 & 0 & \alpha^{-1} \end{bmatrix}$$

The next generation matrix is obtained as:

$$FV^{-1} = \begin{bmatrix} \frac{\pi_C \rho \, \beta_1}{(\omega_1 + \mu_C)^2 \, \alpha} & 0 & 0 & \frac{\beta_1 \pi_C}{(\omega_1 + \mu_C) \, \alpha} \\ \frac{\pi_L \beta_2}{\mu_L \, (\omega_1 + \mu_C)} & 0 & 0 & 0 \\ \frac{\pi_H \beta_4}{\mu_H \, (\omega_1 + \mu_C)} & \frac{\pi_H \beta_5}{\mu_H \, (\phi_L + \mu_L)} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}.$$
(22)

The basic contamination number, R_0 is obtained by computing the spectral radius (FV^{-1}) of the next generation matrix. Thus, the dominant eigenvalue of matrix (22) gives the basic contamination number as Eq. (23). In this case, R_0 is defined as number of tonnes of contaminated crops as a result of one tonne of contaminated crops.

$$R_0 = \text{Spectral radius of } FV^{-1} = \frac{\beta_1 \pi_C \rho}{\left[\omega_1 + \mu_C\right]^2 \alpha}$$
(23)

It can be seen that

$$R_0 = R_{eA} \cdot R_{eC}$$

where

$$\begin{split} R_{0A} &= \beta_1 S_C^0 \cdot \frac{1}{\omega_1 + \mu_C}, \\ R_{0C} &= \rho \cdot \frac{1}{\alpha}. \end{split}$$

The basic contamination number, R_0 is analogous to basic reproduction number in epidemiological models, a threshold quantity used to examine equilibrium points. It is a crucial quantity that defines how contamination behaves. If $R_0 > 1$, aflatoxi contamination persists and if $R_0 < 1$, aflatoxin contamination diminishes. On R_{0A} , the term $\beta_1 S_C^0$ represent new contamination while $\frac{1}{\omega_1 + \mu_C}$

represent duration of aflatoxin contamination stay in contaminated crops. On R_{0C} , the term ρ represent new aflatoxin fungi in soil from crops while $\frac{1}{\alpha}$ represent duration of stay of aflatoxin fungi in soil. In this case, R_0 is defined as number of tonnes of contaminated crops as a result of one tonne of contaminated crops.

Local stability of contamination free equilibrium point

In this section we investigate the local stability of the aflatoxin contamination free equilibrium point using Routh-Hurwitz criterion.

Theorem 1. The aflatoxin contamination-free equilibrium point of model system (4) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof. We show that all eigenvalues of F - V matrix of model system (4) at contamination free equilibrium have negative real part. Matrices *F* and *V* are defined in Eqs. (20) and (21) while matrix F - V at aflatoxin contamination free is given by:

$$F - V = \begin{bmatrix} -(\omega_1 + \mu_C) & 0 & 0 & \frac{\pi_C \beta_1}{\omega_1 + \mu_C} \\ \frac{\beta_2 \pi_L}{\mu_L} & -(\mu_L + \phi_L) & 0 & 0 \\ \frac{\beta_4 \pi_H}{\mu_H} & \frac{\beta_5 \pi_H}{\mu_H} & -(\mu_H + \phi_H) & 0 \\ \rho & 0 & 0 & -\alpha \end{bmatrix},$$
(24)

The third column of matrix (24) contain diagonal term, it forms an obvious eigenvalue; $\lambda_1 = -(\mu_H + \phi_H)$. Thus the matrix reduces to:

$$F - V = \begin{bmatrix} -(\omega_1 + \mu_C) & 0 & \frac{\pi_C \beta_1}{\omega_1 + \mu_C} \\ \frac{\beta_2 \pi_L}{\mu_L} & -(\mu_L + \phi_L) & 0 \\ \rho & 0 & -\alpha \end{bmatrix}.$$
 (25)

The second column of matrix (25) contain diagonal term also, it form an obvious eigenvalue; $\lambda_2 = -(\mu_L + \phi_L)$. Thus, matrix reduces to:

$$F-V = \begin{bmatrix} -(\omega_1+\mu_C) & \frac{\beta_1\pi_C}{\omega_1+\mu_C} \\ \rho & -\alpha \end{bmatrix}.$$

Taking $|(F - V) - \lambda| = 0$, we have

$$\begin{vmatrix} -(\omega_1 + \mu_C) - \lambda & \frac{\beta_1 \pi_C}{\omega_1 + \mu_C} \\ \rho & -\alpha - \lambda \end{vmatrix} = 0,$$

resulting to the following quadratic equation

$$\lambda^{2} + a_{1}\lambda + a_{2} = 0,$$
(26)
$$(\mu_{c}^{2} + 2\mu_{c}\omega_{1} + \omega_{2}^{2})(1 + \mu_{2})\alpha - a\beta_{1}\pi_{c}(1 - \mu_{1})$$

where $a_1 = [\alpha + \omega_1 + \mu_C]$ and $a_2 = \frac{(\mu_C^2 + 2\mu_C\omega_1 + \omega_1^2)(1 + u_2)\alpha - \rho\beta_1\pi_C(1 - u_1)}{\omega_1 + \mu_C}$.

The necessary and sufficient condition for local stability of the system is that all eigenvalues have negative real parts. It is clear that λ_1 and λ_2 have negative real parts. Using the Routh–Hurwitz criterion, the remaining two eigenvalues have negative real parts if all coefficients of Eq. (26) are greater than zero. Then

$$a_1 = (\omega_1 + \alpha + \mu_C) > 0$$
 and $a_2 = \frac{\alpha \mu_C^2 + 2\alpha \mu_C \omega_1 + \alpha \omega_1^2 - \rho \beta_1 \pi_C}{\omega_1 + \mu_C} = (1 - R_0)\alpha(\omega_1 + \mu_C) > 0$ if $R_0 < 1$. Therefore, the aflatoxin

contamination-free equilibrium point is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Global stability of aflatoxin contamination free equilibrium point

In this subsection we perform global stability analysis of aflatoxin contamination free equilibrium point using the approach explained by [25].

Theorem 2. The aflatoxin contamination free equilibrium point of the aflatoxin contamination model system (4) is globally asymptotically stable on Ω if $R_0 < 1$.

Proof. Model system (4) can be expressed as:

$$\begin{cases} \frac{dX_S}{dt} = A(X_S - X_{DFE,S}) + A_1 X_C, \\ \frac{dX_C}{dt} = A_2 X_C, \end{cases}$$
(27)

where, X_S is the vector representing the compartments that do not transmit aflatoxin and X_C represent aflatoxin contaminating compartments. In case A_2 is a stable Metzler matrix and A has real negative eigenvalues, the aflatoxin contamination free equilibrium is globally asymptotically stable. From the model system (4) it can be deduced that:

$$\begin{split} X_{C} &= (C_{C}, C_{L}, C_{H}, A)^{T}, \quad X_{S} = (S_{C}, S_{L}, S_{H})^{T}, \\ X_{S} - X_{DFE,S} &= \begin{bmatrix} S_{C} - \frac{\pi_{C}}{\omega_{1} + \mu_{C}} \\ S_{L} - \frac{\pi_{L}}{\mu_{L}} \\ S_{H} - \frac{\pi_{H}}{\mu_{H}} \end{bmatrix}, \quad A_{1} = \begin{bmatrix} 0 & 0 & 0 & -\beta_{1}S_{C} \\ -\beta_{2}S_{L} & 0 & 0 & 0 \\ -\beta_{4}S_{H} & -\beta_{5}S_{H} & 0 & 0 \end{bmatrix}. \end{split}$$

Matrix A is obtained from non contaminating classes in system (4) and is given by:

$$A = \begin{bmatrix} -(\omega_1 + \mu_C) & 0 & 0 \\ 0 & -\mu_L & 0 \\ 0 & 0 & -\mu_H \end{bmatrix}.$$

It can be seen that the eigenvalues of matrix A are all negative; $\lambda_1 = -\omega_1 - \mu_C$, $\lambda_2 = -\mu_L$ and $\lambda_3 = -\mu_H$. Matrix A_2 is obtained from contaminating classes in system (4) and is given by:

$$A_{2} = \begin{bmatrix} -(\omega_{1} + \mu_{C}) & 0 & 0 & \frac{\beta_{1}\pi_{C}}{\omega_{1} + \mu_{C}} \\ \frac{\beta_{2}\pi_{L}}{\mu_{L}} & -(\mu_{L} + \phi_{L}) & 0 & 0 \\ \frac{\beta_{4}\pi_{H}}{\mu_{H}} & \frac{\beta_{5}\pi_{H}}{\mu_{H}} & -(\mu_{H} + \phi_{H}) & 0 \\ \rho & 0 & 0 & -\alpha \end{bmatrix}$$

It can be observed that matrix A_2 is the Metzler matrix since its out-diagonal entries are non-negative. To prove the stability of A_2 , we adopt the idea of stable Metzler matrix [26] and apply Lemma 4 in 'Appendix'.

Comparing Metzler matrix A_2 with a Metzler matrix M in Lemma 4, matrices U, V, X and Y are obtained as:

$$\begin{split} U &= \begin{bmatrix} -(\omega_1 + \mu_C) & 0\\ \frac{\beta_2 \pi_L}{\mu_L} & -(\mu_L + \phi_L) \end{bmatrix}, \ V = \begin{bmatrix} 0 & \frac{\beta_1 \pi_C}{\omega_1 + \mu_C} \\ 0 & 0 \end{bmatrix}, \ X = \begin{bmatrix} \frac{\beta_4 \pi_H}{\mu_H} & \frac{\beta_5 \pi_H}{\mu_H} \\ \rho & 0 \end{bmatrix}, \\ Y &= \begin{bmatrix} -(\mu_H + \phi_H) & 0\\ 0 & -\alpha \end{bmatrix}. \end{split}$$

It can be seen clearly that matrix U is stable matrix since all eigenvalues have negative real parts. Upon computation we obtain:

$$Y - XU^{-1}V = \begin{bmatrix} -(\mu_H + \phi_H) & \frac{\pi_H \left(\beta_5 \beta_2 \pi_L + \beta_4 \mu_L^2 + \beta_4 \mu_L \phi_L\right) \beta_1 \pi_C}{\mu_H \mu_L \left(\omega_1 + \mu_C\right)^2 \left(\mu_L + \phi_L\right)} \\ 0 & -(1 - R_0)\alpha \end{bmatrix}$$

 $Y - XU^{-1}V$ is Metzler stable if $R_0 < 1$. Therefore, the aflatoxin contamination free equilibrium point of the model system (4) is globally asymptotically stable if $R_0 < 1$ since matrix A have real negative eigenvalues and Matrix A_2 is a Metzler stable matrix.

Aflatoxin contamination persistence equilibrium (ACPE) point

The aflatoxin contamination persistence equilibrium point $E^*(S_C^*, C_C^*, S_L^*, C_L^*, S_H^*, S_H^*, A^*)$ of the model system (4) is obtained by setting all equations to zero and solving for the state variables. After solving E^* is given by:

$$\begin{split} S_{C}^{*} &= \frac{\pi_{C}}{R_{0}\left(\omega_{1} + \mu_{C}\right)}, \\ C_{C}^{*} &= \frac{Q_{7}(R_{0} - 1)}{Q_{8}}, \\ S_{L}^{*} &= \frac{Q_{1}R_{0}^{2}}{Q_{2}(R_{0} - 1) + Q_{3}R_{0}}, \\ C_{L}^{*} &= \frac{Q_{4}R_{0}(R_{0} - 1)}{Q_{5}(R_{0} - 1) + Q_{6}R_{0}}, \\ S_{H}^{*} &= \frac{\pi_{H}Q_{8}\left(Q_{5}\left(R_{0} - 1\right) + Q_{6}R_{0}\right)}{\beta_{4}\left(Q_{5}\left(R_{0} - 1\right) + Q_{6}R_{0}\right)Q_{7}\left(R_{0} - 1\right) - Q_{8}\left(Q_{9} + Q_{4}R_{0}\left(R_{0} - 1\right)\beta_{5} + \mu_{H}Q_{5}\left(R_{0} - 1\right)\right)}, \end{split}$$

$$C_{H}^{*} = \frac{\left(\beta_{4}\left(Q_{5}\left(R_{0}-1\right)+Q_{6}R_{0}\right)Q_{7}\left(R_{0}-1\right)+Q_{4}R_{0}\left(R_{0}-1\right)\beta_{5}Q_{8}\right)\pi_{H}}{\left(\beta_{4}\left(Q_{5}\left(R_{0}-1\right)+Q_{6}R_{0}\right)Q_{7}\left(R_{0}-1\right)-Q_{8}\left(Q_{9}+Q_{4}R_{0}\left(R_{0}-1\right)\beta_{5}+\mu_{H}Q_{5}\left(R_{0}-1\right)\right)\right)Q_{10}},$$

$$A^{*} = \left(R_{0}-1\right)\left(\omega_{1}+\mu_{C}\right).$$

The denominator of S_H^* can be simplified to

$$\left[\beta_4 Q_6 Q_7 + Q_5 Q_7 - Q_4 Q_5 Q_8\right] (R_0 - 1)^2 + \left[\beta_4 Q_6 Q_7 - Q_5 Q_8 \mu_H\right] (R_0 - 1) - Q_8 (Q_4 + Q_9) + Q_8 Q_8 + Q_9 +$$

Using completing the square technique we obtain the following:

$$\left[(R_0 - 1) - \frac{Q_{13} + Q_5 Q_7 - Q_{14}}{Q_{13} - Q_5 Q_8 \mu_H} \right]^2 - \frac{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right] (Q_8 (Q_4 + Q_9))}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2} \right]^2 - \frac{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_8 \mu_H \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_8 \mu_H \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_8 \mu_H \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_8 \mu_H \right]^2} - \frac{(Q_{13} - Q_5 Q_8 \mu_H)^2}{4 \left[Q_{13} + Q_5 Q_8 \mu_H \right]^2} - \frac{(Q_{13} - Q_{14} \mu_H)^2}{4 \left[Q_{13} + Q_8 \mu_H \right]^2} - \frac{(Q_{13} - Q_{14} \mu_H)^2}{4 \left[Q_{13} + Q_8 \mu_H \right]^2} - \frac{(Q_{13} - Q_{14} \mu_H)^2}{4 \left[Q_{13} + Q_8 \mu_H \right]^2} - \frac{(Q_{13} - Q_{14} \mu_H)^2}{4 \left[Q_{13} + Q_8 \mu_H \right]^2} - \frac{(Q_{13} - Q_{14} \mu_H)^2}{4 \left[Q_{14} + Q_{14} \mu_H \right]^2} - \frac{(Q_{14} - Q_{14} \mu_H)^2}{4 \left[Q_{14} + Q_{14} \mu_H \right]^2}$$

Upon simplification we have:

$$\left[(R_0 - 1) - \frac{Q_{13} + Q_5 Q_7 - Q_{14}}{Q_{13} - Q_5 Q_8 \mu_H} \right]^2 + \frac{Q_8 (Q_4 + Q_9)}{Q_{12} \left[(1 + Q_{11}) + (R_0 - 1)Q_{11} \right]} + \frac{\left[Q_{13} - Q_5 Q_8 \mu_H \right]^2}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2}$$

Similarly, the denominator of C_H^* can be simplified to

$$\left[(R_0 - 1) - \frac{Q_{13} + Q_5 Q_7 - Q_{14}}{Q_{13} - Q_5 Q_8 \mu_H} \right]^2 Q_{10} + \frac{Q_8 (Q_4 + Q_9) Q_{10}}{Q_{12} \left[(1 + Q_{11}) + (R_0 - 1) Q_{11} \right]} + \frac{\left[Q_{13} - Q_5 Q_8 \mu_H \right]^2 Q_{10}}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2} \right]^2 Q_{10} + \frac{Q_8 (Q_4 + Q_9) Q_{10}}{Q_{12} \left[(1 + Q_{11}) + (R_0 - 1) Q_{11} \right]} + \frac{\left[Q_{13} - Q_5 Q_8 \mu_H \right]^2 Q_{10}}{4 \left[Q_{13} + Q_5 Q_7 - Q_{14} \right]^2}$$

where $Q_1 = (\omega_1 + \mu_C)^3 \alpha \pi_L$, $Q_2 = \pi_C^2 \rho \beta_1 \beta_2$, $Q_3 = \pi_C \rho \beta_1 (\mu_C \mu_L + \mu_L \omega_1)$, $Q_4 = \alpha (\omega_1 + \mu_C)^2$, $Q_5 = \pi_C \rho \beta_1 \beta_2 (\phi_L + \mu_L)$, $Q_6 = \rho \beta_1 (\phi_L + \mu_L) (\mu_C \mu_L + \mu_L \omega_1)$, $Q_7 = \alpha \omega_1 + \alpha \mu_C$, $Q_8 = \rho \beta_1$, $Q_9 = R_0 Q_6 \mu_H$, $Q_{10} = (\mu_H + \phi_H)$, $Q_{11} = \frac{(\omega_1 + \mu_C)^2 \alpha}{\pi_C}$, $Q_{12} = Q_5 \alpha (\omega_1 + \mu_C)$, $Q_{13} = \beta_4 Q_6 Q_7$ and $Q_{14} = Q_4 Q_5 Q_8$.

Therefore, the aflatoxin contamination persistence equilibrium point exist if $R_0 > 1$.

Global stability of aflatoxin contamination persistence equilibrium point

Theorem 3. The aflatoxin contamination persistence equilibrium point of the aflatoxin contamination model system (4) is globally asymptotically stable on Ω if $R_0 > 1$.

Proof. A Lyapunov function of model system (4) as describe by [27,28] was employed in this study. The Lyapunov function *H* is defined by

$$H = \sum P_i \left(y_i - y_i^* - y_i^* \ln \frac{y_i}{y_i^*} \right)$$

where P_i denotes a positive constant to be determined, y_i denotes a population of *i*th compartment and y_i^* denotes an aflatoxin contamination-persistence equilibrium point of the model. It is clear that the function *H* satisfy all conditions for Lyapunov function as follows:

- (i) *H* is zero at the equilibrium $E^*(S_C^*, C_C^*, S_L^*, C_L^*, S_H^*, A^*)$
- (ii) *H* is positive for all other values of $S_C, C_C, S_L, C_L, S_H, S_H$ and *A*.

Note that E^* exist only if ($R_0 > 0$). Consequently, H satisfies conditions for Lyapunov function only if ($R_0 > 0$). A Lyapunov function H of the model system (4) is defined by

$$H = P_1(S_C - S_C^* - S_C^* \ln \frac{S_C}{S_C^*}) + P_2(C_C - C_C^* - C_C^* \ln \frac{C_C}{C_C^*}) + P_3(S_L - S_L^* - S_L^* \ln \frac{S_L}{S_L^*}) + P_4(C_L - C_L^* - C_L^* \ln \frac{C_L}{C_L^*}) + P_5(S_H - S_H^* - S_H^* \ln \frac{S_H}{S_H^*}) + P_6(C_H - C_H^* - C_H^* \ln \frac{C_H}{C_H^*}) + P_7(A - A^* - A^* \ln \frac{A_{A^*}}{A^*}).$$
(28)

where P_1 , P_2 , P_3 , P_4 , P_5 , P_6 and P_7 are positive constants to be determined. The derivative of the Lyapunov function H with respect to time is given by

$$\frac{dH}{dt} = P_1 \left(1 - \frac{S_C^*}{S_C} \right) \frac{dS_C}{dt} + P_2 \left(1 - \frac{C_C^*}{C_C} \right) \frac{dC_C}{dt} + P_3 \left(1 - \frac{S_L^*}{S_L} \right) \frac{dS_L}{dt} + P_4 \left(1 - \frac{C_L^*}{C_L} \right) \frac{dC_L}{dt} + P_5 \left(1 - \frac{S_H^*}{S_H} \right) \frac{dS_H}{dt} + P_6 \left(1 - \frac{C_H^*}{C_H} \right) \frac{dC_H}{dt} + P_7 \left(1 - \frac{A^*}{A} \right) \frac{A}{dt}.$$
(29)

(30)

Substituting $\frac{dS_C}{dt}$, $\frac{dC_C}{dt}$, $\frac{dS_L}{dt}$, $\frac{dC_L}{dt}$, $\frac{dH_H}{dt}$, $\frac{dC_H}{dt}$ and $\frac{dA}{dt}$ in Eq. (29) it yields $\frac{dH}{dt} = P_1 \left(1 - \frac{S_C^*}{S_C} \right) \left[\pi_C - \beta_1 A S_C - k_1 S_C \right]$ + $P_2\left(1-\frac{C_C^*}{C_C}\right)\left[\beta_1AS_C-k_1C_C\right]$ + $P_3\left(1-\frac{S_L^*}{S_L}\right)\left[\pi_L-\beta_2C_CS_L-\mu_LS_L\right]$ + $P_4\left(1-\frac{C_L^*}{C_L}\right)\left[\beta_2 C_C S_L - k_2 C_L\right]$ + $P_5\left(1-\frac{S_H^*}{S_H}\right)\left[\pi_H-\beta_4C_CS_H-\beta_5C_LS_H-\mu_HS_H\right]$ + $P_6\left(1-\frac{C_H^*}{C_H}\right)\left[\beta_4 C_C S_H + \beta_5 C_L S_H - k_3 C_H\right]$ $+ P_7 \left(1 - \frac{A^*}{A}\right) \left[\rho C_C - \alpha A\right],$

where $k_1 = \omega_1 + \mu_C$, $k_2 = \mu_L + \phi_L$ and $k_3 = \mu_H + \phi_H$. At aflatoxin contamination-persistence equilibrium point E^* Eq. (30) yields:

$$\frac{dH}{dt} = P_{1} \left(1 - \frac{S_{C}^{*}}{S_{C}} \right) \left[\beta_{1}AS_{C}^{*} + k_{1}S_{C}^{*} - \beta_{1}AS_{C} - k_{1}S_{C} \right]
+ P_{2} \left(1 - \frac{C_{C}^{*}}{C_{C}} \right) \left[\beta_{1}AS_{C} - \frac{\beta_{1}A^{*}S_{C}^{*}C_{C}}{C_{C}^{*}} \right]
+ P_{3} \left(1 - \frac{S_{L}^{*}}{S_{L}} \right) \left[\beta_{2}C_{C}S_{L}^{*} + \mu_{L}S_{L}^{*} - \beta_{2}C_{C}S_{L} - \mu_{L}S_{L} \right]
+ P_{4} \left(1 - \frac{C_{L}^{*}}{C_{L}} \right) \left[\beta_{2}C_{C}S_{L} - \frac{\beta_{2}C_{C}^{*}S_{L}^{*}C_{L}}{C_{L}^{*}} \right]
+ P_{5} \left(1 - \frac{S_{H}^{*}}{S_{H}} \right) \left[\beta_{4}C_{C}^{*}S_{H}^{*} + \beta_{5}C_{L}^{*}S_{H}^{*} + \mu_{H}S_{H}^{*} - \beta_{4}C_{C}S_{H} - \beta_{5}C_{L}S_{H} - \mu_{H}S_{H} \right]
+ P_{6} \left(1 - \frac{C_{H}^{*}}{C_{H}} \right) \left[\beta_{4}C_{C}S_{H} + \beta_{5}C_{L}S_{H} - \frac{\beta_{4}C_{C}^{*}S_{H}^{*}C_{H}}{C_{H}^{*}} - \frac{\beta_{5}C_{L}^{*}S_{H}^{*}C_{H}}{C_{H}^{*}} \right]
+ P_{7} \left(1 - \frac{A^{*}}{A} \right) \left[\alpha A^{*} - \rho C_{C}^{*} + \rho C_{C} - \alpha A \right].$$
(31)

For simplification, let $e = \frac{S_C}{S_C^*}$, $f = \frac{C_C}{C_C^*}$, $g = \frac{S_L}{S_I^*}$, $h = \frac{C_L}{C_I^*}$, $m = \frac{S_H}{S_H^*}$, $n = \frac{C_H}{C_H^*}$ and $q = \frac{A}{A^*}$. Upon simplification, Eq. (31) yields;

$$\begin{aligned} \frac{dH}{dt} &= -P_1 k_1 S_C \left(1 - \frac{1}{e}\right)^2 - P_3 \mu_L S_L \left(1 - \frac{1}{g}\right)^2 - P_5 \mu_H S_H \left(1 - \frac{1}{m}\right)^2 - P_7 \alpha A \left(1 - \frac{1}{q}\right)^2 \\ &+ (P_1 + P_2) \beta_1 A^* S_C^* + (P_3 + P_4) \beta_2 C_C * S_L * + (P_5 + P_6) \left[\beta_4 C_C^* S_H^* + \beta_5 C_L^* S_H^*\right] - P_7 \rho C_C^* \\ &- P_1 \beta_1 A^* S_C^* \cdot \frac{1}{e} + \beta_1 A^* S_C^* (P_2 - P_1) \cdot eq + P_1 \beta_1 A^* S_C^* \cdot q \\ &+ \left(P_3 \beta_2 C_C * S_C^* + P_7 \rho C_C^* - P_2 \beta_1 A^* S_C^* + P_5 \beta_4 C_C^* S_H^*\right) \cdot f - P_2 \beta_1 A^* S_C^* \cdot \frac{eq}{f} \end{aligned}$$
(32)
$$&+ \beta_2 C_C^* S_L^* (P_4 - P_3) \cdot f g - P_3 \beta_2 C_C^* S_L^* \cdot \frac{1}{g} + \left(P_5 \beta_5 C_L S_H^* - P_4 \beta_2 C_C^* S_L^*\right) \cdot h \\ &- P_4 \beta_2 C_C^* S_L^* \cdot \frac{fg}{h} + (P_6 - P_5) \beta_4 C_C^* S_H^* \cdot f m + (P_6 - P_5) \beta_4 C_L^* S_H^* + hm \\ &- P_5 \left(\beta_4 C_C^* S_H^* + \beta_5 C_L^* S_H^*\right) \cdot \frac{1}{m} - P_6 \left(\beta_4 C_C^* S_H^* + \beta_5 C_L^* S_H^*\right) \cdot n - P_6 \beta_4 C_C^* S_H^* \cdot \frac{fm}{n} \\ &- P_6 \beta_5 C_L^* S_H^* \cdot \frac{hm}{n} - P_7 \rho C_C^* \cdot \frac{f}{q} + P_7 \rho C_C^* \cdot \frac{1}{q} \end{aligned}$$

Setting the coefficients of eq, fg, h, fm and hm be equal to zero, we have $P_1 = P_2 = P_3 = P_4 = P_7 = 1$ and $P_5 = P_6 = \frac{\beta_2 C_c^* S_L^*}{\beta_5 C_c^* S_T^*}$ Eq. (32) can be written as;

$$\frac{dH}{dt} = -P_1 k_1 S_C \left(1 - \frac{1}{e}\right)^2 - P_3 \mu_L S_L \left(1 - \frac{1}{g}\right)^2 - P_5 \mu_H S_H \left(1 - \frac{1}{m}\right)^2 - P_7 \alpha A \left(1 - \frac{1}{q}\right)^2 + \beta_1 A^* S_C^* \left(2 - \frac{1}{e} + q - f - \frac{eq}{f}\right) + \beta_2 C_C^* S_L^* \left(4 + f - \frac{1}{g} - \frac{fg}{h} - \frac{1}{m} - n - \frac{hm}{n}\right)$$
(33)

Table 1

Description of parameters used in numerical simulations.

| Parameter | Description | Unit | Value | Source |
|------------|---|---|---------|----------|
| β_1 | Aflatoxin contamination rate of crops | (A. fungi \times year) ⁻¹ | 0.05 | [19] |
| β_2 | Aflatoxin contamination rate of livestock from crops | $(Crops \times year)^{-1}$ | 0.003 | [22] |
| β_4 | Aflatoxin contamination rate of humans from crops | $(Crops \times year)^{-1}$ | 0.002 | [22] |
| β_5 | Aflatoxin contamination rate of humans from livestock | $(Livestock \times year)^{-1}$ | 0.001 | [22] |
| ρ | Shading rate of aflatoxin fungi from crops to soil | A. fungi \times (Crops \times year) ⁻¹ | 0.019 | [1] |
| π_{C} | Crops production rate | $Crops \times year^{-1}$ | 180,000 | See text |
| π_L | Recruitment rate of livestock | Livestock \times year ⁻¹ | 255,000 | See text |
| π_H | Recruitment rate of humans | Humans \times year ⁻¹ | 37,500 | See text |
| μ_L | Natural death rate of livestock | year ⁻¹ | 0.17 | See text |
| μ_H | Natural death rate of humans | year ⁻¹ | 0.015 | See text |
| ϕ_L | Livestock death rate due to aflatoxicosis | year ⁻¹ | 0.2 | [35] |
| ϕ_H | Human death rate due to aflatoxicosis | year ⁻¹ | 0.1 | [8] |
| ω_1 | Consumption rate of crops | year ⁻¹ | 0.75 | [23] |
| μ_C | Loss rate of crops | year ⁻¹ | 0.25 | See text |
| α | Reduction rate of aflatoxin fungi in soil | vear ⁻¹ | 0.1 | [22] |

$$\frac{\beta_2 \beta_4 C_C^* S_L^*}{\beta_5 C_L^*} \left(2 + f - \frac{1}{m} - n - \frac{fm}{n} \right) + \rho C_C^* \left(f - 1 - \frac{f}{q} + \frac{1}{q} \right)$$

Consider a function $z(x) = 1 - x + \ln(x) \le 0$ for any x > 0 with equality holds if x = 1. Thus $1 - x \le -\ln(x)$. Taking:

$$2 - \frac{1}{e} + q - f - \frac{eq}{f} = \left(1 - \frac{1}{e}\right) + (1 - f) + \left(1 - \frac{eq}{f}\right) - (1 - q) \le -\ln(\frac{1}{e}) - \ln(f) - \ln\left(\frac{eq}{f}\right) + \ln(f) = 0.$$

$$4 + f - \frac{1}{g} - \frac{fg}{h} - \frac{1}{m} - n - \frac{hm}{n} = \left(1 - \frac{1}{g}\right) + \left(1 - \frac{fg}{h}\right) + \left(1 - \frac{1}{m}\right) + (1 - n) + \left(1 - \frac{hm}{n}\right) - (1 - f)$$

$$\le -\ln\left(\frac{1}{g}\right) - \ln\left(\frac{fg}{h}\right) - \ln\frac{1}{m} - \ln(m) - \ln\left(\frac{hm}{n}\right) + \ln(f) = 0.$$

$$2 + f - \frac{1}{m} - n - \frac{fm}{n} = \left(1 - \frac{1}{m}\right) + (1 - n) + \left(1 - \frac{fm}{n}\right) - (1 - f)$$

$$\le -\ln\left(\frac{1}{m}\right) - \ln(n) - \ln\left(\frac{fm}{n}\right) + \ln(f) = 0.$$

$$f - 1 - \frac{f}{q} + \frac{1}{q} = -(1 - f) + \left(1 - \frac{f}{q}\right) - \left(1 - \frac{1}{q}\right) \le \ln(f) - \ln\left(\frac{f}{g} + \ln\left(\frac{1}{q}\right)\right) = 0$$

Thus, $\frac{dH}{dt} \leq 0$. Using LaSalle's extension to Lyapunov's method, the limit set of each solution is contained in the largest invariant

set for which $S_C^* = S_C$, $C_C^* = C_C$, $S_L^* = S_L$, $C_L^* = C_L$, $S_H^* = S_H$, $C_H^* = C_H$, $A^* = A$ which is the singleton $\{E^*\}$ [29]. Hence, the aflatoxin contamination-persistence equilibrium point (E^*) of the model system (4) is global asymptotically stable on Ω when $R_e > 1$. The global asymptotic stability of aflatoxin contamination-persistence equilibrium point suggests that contamination will persist at a relatively constant level in the population, without dying out or causing a large-scale aflatoxin contamination.

Numerical simulation

Model parameters and initial conditions

The values of parameters used were based on humans, cattle for livestock and maize for crops as shown in Table 1. For numerical simulation purposes, we have taken data from the Dodoma region in Tanzania, which is estimated to have 2,500,000 people and 1,500,000 cattle [30,31]. The life span of humans in Tanzania is about 66 years [30]. Thus, death rate of humans is estimated to be $\mu_H = \frac{1}{66} = 0.015$, while the recruitment rate is estimated as, $\pi_H = 2,500,000 \times 0.015 = 37,500$. On the other hand, the life span of cattle is estimated to be 6 years [32], therefore we can estimate the death rate of cattle to be $\mu_L = \frac{1}{6} = 0.17$ and the recruitment rate $\pi_H = 1,500,000 \times 0.17 = 255,000$. Furthermore, it is estimated that Dodoma region produces 180,000 tonnes of maize per year [33] while 25% of it is lost during or after harvest [34]. Therefore, we estimate the maize recruitment rate, $\pi_C = 180,000$, maize loss rate, $\mu_C = 0.25$ and the maize consumption rate, $\omega_1 = 0.75$ assuming that all maize produced are consumed within a year. The initial values used in this study are: [$S_C(0) C_C(0) S_L(0) C_L(0) S_H(0) C_H(0) A(0)$] = [720,000 0 1,5000,000 0 2,500,000 0 18000]. Prior to performing numerical simulations, we show sensitivity analyses of all parameters with respect to model variables in Section "Sensitivity Analysis".



Fig. 2. Sensitivity analysis PRCCs.

Sensitivity analysis

Global sensitivity analysis of parameters with respect to all variables was performed using the Latin Hypercube Sampling (LHS) approach as explained by [36]. We also compute the Partial Rank Correlation Coefficients (PRCCs) of the model parameters with respect to the state variables to assess whether their uncertainties have a significant contribution using the approach described by [36,37]. The value of PRCC indicates the influence of a parameter on the state variable. A value approaching 1 or -1 indicates that a parameter has greater influence, while values between 0.3 and -0.3 indicate weak influence. The sign of PRCC shows how a parameter influences the state variable, with positive values indicating positive influence and negative values indicating negative influence. All parameters except the recruitment and death rates are included in the sensitivity analysis. In the following subsection, we report sensitivity results on aflatoxin fungi, contaminated crops, contaminated livestock and contaminated humans.

Sensitivity of parameters on aflatoxin fungi

The time-variable PRCCs for aflatoxin fungi are shown in Fig. 2(a). It can be seen that the PRCC for aflatoxin fungi shading rate (ρ) has the most positive value (approaching 1), meaning that it has a strong influence and its increase leads to an increase in aflatoxin fungi in soil and vice versa. Crop consumption (ω_1) and the reduction rate of aflatoxin fungi in soil (α) have the most negative values (approaching -1) as shown by Fig. 2(a), meaning that when they increase, aflatoxin fungi in soil decrease.

Sensitivity of parameters on contaminated crops

The time-variable PRCCs for contaminated crops are shown in Fig. 2(b). The crop consumption rate (ω_1) has the most negative value and it falls out of the shaded region (between 3 and -3) as shown by 2(b) meaning that its increase leads to a decrease in contaminated crops and vice versa. The crop contamination rate (β_1) has PRCCs above 0.3 on the first year of simulation, indicating a strong positive influence at the beginning. It also indicates that any control measure to reduce crop contamination rate should be strongly applied in the first years. Applying control measures to reduce crop contamination rate after the first year will have low efficiency.



Fig. 3. Convergence to equilibrium point with different initial values when $R_0 < 1$ and when $R_0 > 1$.

Sensitivity of parameters on contaminated livestock

The time-variable PRCCs for contaminated livestock are shown in Fig. 2(c). The crop consumption rate (ω_1) has the most negative value, as shown by 2(c), meaning that when it increases, contaminated livestock decreases. Livestock contamination rate (β_2) has the most positive value, meaning that its increase leads to an increase in contaminated livestock and vice versa. It can be seen that the PRCCs for livestock contamination rate (β_2) crosses 0.3 after the first year, indicating that any control measure on it will have high efficiency after the first year.

Sensitivity of parameters on contaminated humans

The time-variable PRCCs for contaminated humans are shown in Fig. 2(d). It can be seen that PRCCs for all parameters are fluctuating within the weak region (-3 to 3), with humans contamination rate from crops (β_4) and from livestock (β_5) crossing out



Fig. 4. Population dynamics for all compartments.

at some points. Crop contamination rate (β_1) and livestock contamination rate (β_4) have more influence on the first year, suggesting the need for early control measures.

Numerical results

We start by showing the behavior of solution trajectories within different planes plotted from different initial points. The solution trajectories with different initial points in the $S_C C_C C_H$, $S_C S_L S_H$ and $A C_C C_L$ -planes are respectively shown in Figs. 3(a)–3(c). When $R_0 < 1$, all trajectories that start inside the region of attraction approach the aflatoxin contamination-free equilibrium (E^0) point. When $R_0 > 1$, the solution trajectories with different initial points in the $S_C C_C C_H$, $S_L C_L C_H$ and $A S_H C_H$ -planes are respectively shown Figs. 3(d)–3(f). In this case, all trajectories that start in the region of attraction approach the aflatoxin contamination persistence equilibrium (E^*) point.

Fig. 4 shows the dynamics of crops, livestock, humans and aflatoxin fungi populations with time. It further demonstrates a decrease in susceptible humans and a rapid increase in contaminated humans, which at some point starts to decrease due to contamination-induced and natural death. This trend has also been reported by [38], that in Africa there is highest exposure leading to higher rate of aflatoxin contamination. Contaminated livestock experience the same trend: a rapid increase at the initial stages but a decrease at some point due to contamination-induced and natural death. Aflatoxin-producing fungi have shown an increase at the beginning before reaching equilibrium over time.

The values in Table 1 were used as baseline parameter values. Fig. 5 shows that, using baseline parameters, the contamination number is $R_0 = 9.5 > 1$ meaning that contamination persists in the population. Our aim was to find a set of parameters with which the aflatoxin contamination diminishes in the population ($R_0 < 1$). Sensitivity analysis results in Section "Sensitivity Analysis" showed that aflatoxin fungi shading (ρ), death of aflatoxin fungi (α), crops contamination (β_1), crops consumption (ω_1), livestock contamination (β_2) and human contamination (β_4 and β_5) rates have more influence on contaminated compartments. However, literature shows that it is difficult to control aflatoxin contamination in livestock and humans [39]. Therefore, we exclude livestock



Fig. 5. Impacts of decreasing β_1 , ρ and increase α on R_0 and sub-populations while other parameters are held constant.

and humans contamination rates (β_2 , β_4 and β_5). Also, based on the fact that maize is the main staple food in the Dodoma region, we exclude the crop consumption rate (ω_1) from the set.

The study evaluated the impact of changes in the rates of crops contamination (β_1), death (α) and shading of aflatoxin fungi in soil (ρ) on the basic contamination number R_0 and contamination dynamics. It was found that decrease in crops contamination (β_1), shading rate of aflatoxin fungi in soil (ρ) by 50% and increase in death rate of aflatoxin fungi (α) by 50%, reduce the aflatoxin contamination number to $R_0 = 0.7 < 1$ as shown in Fig. 5 meaning that contamination diminishes in the population. This implies that β_1 , ρ and α have significant impact on controlling aflatoxin contamination. These results suggest that control measures should be applied to crops contamination (β_1), shading (ρ) and death rates of aflatoxin fungi in soil (α) to reduce contamination in the population. Further, results suggest that any control measure that can reduce crops contamination (β_1), shading of aflatoxin fungi in soil rates (ρ) and increase death rate of aflatoxin fungi (α) by 50% will reduce aflatoxin contamination in the population by above 92%.

Conclusion

In this paper, we considered an aflatoxin contamination dynamics model incorporating crops, livestock, humans and aflatoxin fungi populations. Both the aflatoxin contamination free equilibrium (ACFE) and aflatoxin contamination persistence equilibrium (ACPE) points were analyzed, and some criteria were derived to ensure the stability of these equilibrium points. The basic contamination number R_0 was obtained. When $R_0 < 1$, the ACFE is globally asymptotically stable, whereas when $R_0 > 1$ the ACFE is globally asymptotically stable. Partial Rank Correlation Coefficients (PRCCs) for global sensitivity analysis were calculated using Latin Hypercube Sampling (LHS) to see how sensitive and significant the parameter is on each variable. Results from numerical simulation showed that decreasing crop contamination rate (β_1), shading rate (ρ) and increasing death rate of aflatoxin fungi in soil by 50% can reduce the contamination number R_0 from 9.5 to 0.7 (above 92%) meaning that contamination diminishes in the population. The study recommends that control measures be applied to the crop contamination rate (β_1), shading rate (ρ) and death rate of aflatoxin fungi in soil (α) to reduce contamination in the population. Compared to other studies in aflatoxin contamination, the current study provides a thoroughly global sensitivity analysis of parameters involved in contamination and indicated the most important ones for control strategies. Results contributes to implementation of first three sustainable development goal (SDGs) by 2030. No poverty, zero hunger and good health and well-being are directly related to production of food and feeds. As some crops are destroyed each year due to aflatoxin, people suffer extremely hunger as well as incurring losses which accelerate poverty. In some cases, where people and livestock consume crops with above tolerable levels of aflatoxin, they suffer health related impacts including acute aflatoxicosis, liver cancer and immunity suppression. By 2030, the first goal, target 1.1 of SDG aims to eradicate extreme poverty for all people everywhere. Since, majority of people in African countries rely on agriculture as main economic activity, solving aflatoxin contamination problem is vital to achieving this target. By 2030, the second goal, target 2.1 of SDG aims to end hunger and ensure access to safe and nutritious food. Increasing aflatoxin contamination trends in food crops is threatening achievement of this target. Results from this study will enlighten efforts in achieving this target. By 2030, the third goal, target 3.9 of SDG aims to reduce deaths and illness from hazardous chemicals and contamination. As aflatoxin contamination is one of major causes of liver cancer, results from this study aims to provide stakeholders with supporting evidences in preparing control strategies.

CRediT authorship contribution statement

F.A. Mgandu: Conceptualization, Methodology, Writing – original draft, Writing – review & editing. **S. Mirau:** Conceptualization, Methodology, Writing – review & editing, Supervision. **N. Nyerere:** Conceptualization, Methodology, Writing – review & editing, Supervision. **E. Mbega:** Biological significance of the model and findings, Review & editing. **F. Chirove:** Conceptualization, Methodology, Writing – review & editing, Supervision.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Filimon Abel Mgandu reports financial support was provided by Tanzania Ministry of Agriculture under Tanzania Initiative for Preventing Aflatoxin Contamination (TANIPAC).

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Appendix

Lemma 4. Let M be a square Metzler matrix written in block form:

$$M = \begin{bmatrix} U & V \\ X & Y \end{bmatrix}$$

where U and Y are square matrices. M is Metzler stable if and only if matrices U and $Y - XU^{-1}V$ are Metzler stable [40].

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