Falciparum malaria transmission in Masvingo district, Zimbabwe: a mathematical model with varying human and mosquito populations

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ABSTRACT

Introduction: Malaria significantly and disproportionately affects people`s quality of life especially the underprivileged and vulnerable. It poses a serious public health threat despite prevention and control efforts. Masvingo district is not spared, hence, the need to develop a falciparum malaria model and identify the existence of the disease-free equilibrium, analyze its stability, and simulate the model, in the wake of the World Health Organisation Global Malaria Target on local elimination. **Methods:** Deterministic, ordinary differential equation-based SEIR and SEI compartmentalization were used for dynamic human and mosquito populations, respectively. Fewer humans were infected at any given moment due to intervention efforts and there was a lower probability of transmission from a recovered compared to an infected human. The district of Masvingo was purposively chosen, and DHIS2 malaria data from January 2015 through December 2020 was used. **Results:** Following parameter estimation in Microsoft Excel 2019 the disease-free equilibrium point was shown to exist with $R_{0hv}<1$ indicating that it is locally asymptotically stable. It was noted that the dynamics of malaria transmission was variable, while the incidence was generally declining. **Conclusions:** A falciparum malaria model was developed with dynamic human and mosquito populations incorporating recruitment rates, temporary immunity and transmission from recovered humans. The Disease-Free Equilibrium point exists and was shown to be locally asymptotically stable, hence disease will die out suggesting the district is on track to malaria elimination target. To sustain this trajectory a multi-faceted approach is required, including promoting behavior change communication, accessible rapid diagnostic tests and foster collaborations.

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Introduction

Malaria continues to pose a significant worldwide threat to livelihoods and public health, especially in low-to-middle-income nations. It is ranked among the top five infectious disease-related causes of death worldwide, along with respiratory infections, HIV/AIDS, diarrheal illnesses, and tuberculosis, despite decades of interventions [1]. The global estimate of malaria cases in 2023 was at around 263 million associated with an incidence of 60.4 cases per 1000 population at risk, an increase from 58.6 cases per 1000 population at risk in 2022 [2]. In 2020, the World Health Organisation (WHO) estimated that 3.5 billion people were at malaria risk in 87 endemic countries [3]. Over the years, there has been success in lowering death rates; but, the disease burden is still huge and is steadily increasing in many areas, most notably the WHO African Region as well as the WHO Eastern Mediterranean Region [4]. Poor and marginalized people are frequently the most affected, having little access to medical care and preventative measures [1], [2], [3], [4], . The WHO Global Technical Strategy for Malaria highlights the need for better coordinated and strengthened efforts to battle the illness, as the existing global response to malaria is falling short of the targets established for 2025 and 2030 [3].

Controlling the spread of malaria has been a major focus of effort since ancient times, with the ultimate goal being to lessen local transmission and finally eradicate the illness. Reducing morbidity and mortality by 50% was the main goal of malaria control initiatives between 2000 and 2015 [5]. One of the main goals of the Zimbabwe National Health Strategy [6] was to lower the incidence of malaria from 39 cases per 1,000 people in 2014 to 5 cases per 1,000 people by 2020, and to almost completely eliminate malaria-related deaths by the same year. In spite of these lofty objectives, the national incidence rate of malaria stayed at 29 cases per 1,000 population-at risk (PAR) in 2017, declining to 19 cases per 1,000 PAR in 2018, and then somewhat rising to 22 cases per 1,000 PAR in 2019 [6]. As part of a larger plan to eradicate malaria, this raises questions about Zimbabwe's capacity to achieve its current targets of reducing malaria incidence by 75% by 2025 and by 90% by 2030 [2], [3].. To accomplish these goals, more effective and long-lasting interventions are required, according to recent studies.

The World Health Organization reported that for ten high burden African countries, there was an increase of 3.5 million cases in 2017 over the prior year [7]. In the same report, however, Zimbabwe recorded 0.6 million fewer malaria cases in 2018 than in 2017. Despite this decline, Zimbabwe remains a high burden malaria country with about thirty of the sixty-two districts classified as heavy burden malaria districts. For low-burden malaria districts (incidence rate < 5 cases per 1000 PAR) Zimbabwe initiated elimination activities in 20 districts by 2015 and as of 2019 the districts implementing a package of elimination interventions expanded to 29 [8]. These elimination intervention packages include case management, case investigation and classification, and foci investigation and classification, among others. Three of the low-burden malaria districts (Gutu, Chivi and Masvingo) in Masvingo province are being targeted for malaria elimination.

Visual analysis of Masvingo provincial DHIS2 malaria data from 2014 to 2019 identified Masvingo district as a target district for analysis and modelling. In 2018, the district recorded an incidence rate of 2-4 cases per 1000 [8]. The district started implementing elimination packages in 2018 and is one of the target districts for disease elimination by 1. Although there has been some reduction in malaria incidence, but with the objective to locally eliminate the disease, the cyclical nature of the incidence pattern remains of major concern. Hence the necessity to mathematically model the falciparum malaria transmission dynamics in variable human and mosquito populations and assess the implications this has on prevention and control of the disease.

Several authors have developed mathematical models to understand host-vector interactions, transmission dynamics and control measures for local malaria elimination [9], [10], [11]. Compartmental SEIR models have often been used to capture such transmission dynamics between humans and mosquitoes incorporating factors such as newborns and loss in immunity among the recovered population [11]. However, they did not consider re-infection of the recovered population. Research has also highlighted the importance of seasonality and heterogeneity [12], [13],[14],[15] migration and immunity [16], optimal times for interventions as well as the need for micro-models to address challenges associated with local variations [17].

Although not all disease characteristics can be explained by a single model, consideration of these factors enhance a data-driven understanding of disease transmission dynamics and intervention efficacies [18].

The current study was aimed to develop a mathematical model for falciparum malaria transmission dynamics in Masvingo district, assessing the implications this has for achieving the WHO 2030 malaria elimination targets. The study also sought to determine the existence and stability of the disease-free equilibrium and to simulate the model using local malaria data. Thus such modeling as this study provides data-driven insights to support and develop tailored policies and strategies that are geographically appropriate and contextually relevant to malaria prevention and control. Thus, it helps track progress on elimination and understand the impact of the current controls on incidence; hence, guide future decisions such to track progress towards local elimination targets.

Methods

Approach and model formulation

A population-based deterministic approach was used to model the transmission of malaria. The mathematical model consisted of variable human and mosquito populations. The human and mosquito populations were divided into compartments. Infection dynamics in humans were described by ordinary differential equations and used the epidemiological compartment modelling approach as follows: individuals start life being *s*usceptible to falciparum malaria infection (S_h); and these are then exposed at a certain probability to infectious mosquito bites. The *e*xposed (E_h) compartment consists of individuals who are infected but are not capable of infecting others during a latent period. The *i*nfectious humans (I_h) give rise to more infected mosquitoes through their interaction with the susceptible mosquitoes. Once treated, individuals *r*ecover (R_h) from the disease with partial immunity and after some time they become susceptible again (*SEIR*). Although the recovered are not at an immediate risk of developing clinical disease, however, they can still infect susceptible mosquitoes but at a lower rate than that of infectious individuals [21].

Infection dynamics in mosquito vectors followed the *SEI* pattern of compartimentalisation: *s*usceptible mosquitoes (S_v) get infected at a certain probability when they get a blood meal either from infectious humans or recovered humans; then move to *e*xposed compartment (E_v) and finally the *i*nfectious compartment (I_v). The recovered class is here omitted for the mosquito population because once infected the mosquito remains so until death.

The adapted SEIR model was based on Ngwa and Shu [11]. The SEIR model of compartmentalization (figure 1) was chosen because it is more realistic as it includes the latent phase (exposed class) which is not found in SIR or SIS models.

In the Ngwa and Shu model [11], humans follow an SEIR pattern while mosquitoes follow the SEI pattern of compartmentalization. Infectious humans either recover with or without immunity; and those who recover with partial immunity remain infectious while those who recover without immunity return directly to the susceptible compartment. The modeling approach adopted in this research is a modification of the Ngwa and Shu model capturing variable human and mosquito populations, preventive intervention measures such as Long-Lasting Insecticidal Nets (LLINS), immigration, recovery with temporary immunity and

disease-induced death in humans. The direct infectious-to-susceptible transfer that the Ngwa and Shu model contains is excluded from the model, because most people show some varying periods of immunity before becoming susceptible again [22]. Interaction between the two populations is as shown in Figure 1.

Description of model parameters

With reference to Figure 1, Table 1 provides descriptions of the parameters used in the malaria model.

Model assumptions

- The populations under study are heterogeneous and varying with time,
- Once infected, mosquitoes remain so until death,
- The force of infection for humans is not proportional to the average density of infectious vector capacity, but a lower proportion is infected at any given exposure due to the presence of preventive interventions such as LLINs;
- The probability of transmitting infection to susceptible mosquitoes by infectious humans is greater than that by partially immunity recovered humans,
- A person/ mosquito can die at any stage due to natural causes;
- A person once infected can progress to the recovered class or can die from the disease
- Humans recover with temporary immunity, and so can re-enter the susceptible compartment.
- Mosquito dispersal, or migration from surrounding districts is negligible.

Model equations

In view of the model assumptions above and the schematic diagram in Figure 1, the dynamics of malaria transmission was described by the following model equations:

$$\frac{dS_h}{dt} = \Pi_h + \Lambda_h + \rho_h R_h - \frac{\phi\beta_h S_h I_v}{N_h} - \mu_h S_h;\ 0 < \phi < 1 \quad (.1)$$

$$\frac{dE_h}{dt} = \frac{\phi\beta_h S_h I_v}{N_h} - (\alpha_h + \mu_h)E_h; \quad (.2)$$

$$\frac{dI_h}{dt} = \alpha_h E_h - (\mu_h + \delta_h + \lambda_h)I_h \quad (.3)$$

$$\frac{dR_h}{dt} = \lambda_h I_h - (\mu_h + \rho_h)R_h \quad (.4)$$

$$\frac{dS_v}{dt} = \Lambda_v - \frac{\beta_v S_v I_h}{N_v} - \frac{c\beta_v S_v R_h}{N_v} - \mu_v S_v;\ 0 < c < 1 \quad (.5)$$

$$\frac{dE_v}{dt} = \frac{\beta_v S_v I_h}{N_v} + \frac{c\beta_v S_v R_h}{N_v} - (\mu_v + \gamma_v)E_v \quad (.6)$$

$$\frac{dI_v}{dt} = \gamma_v E_v - \mu_v I_v \quad (.7)$$

From the model equations (.1) to (.4) the total population size, N_h, at time *t* is given by:

$$N_h(t) = S_h(t) + E_h(t) + I_h(t) + R_h(t) \quad (.8)$$

This then implies that: $\frac{dN_h}{dt} = \frac{dS_h}{dt} + \frac{dE_h}{dt} + \frac{dI_h}{dt} + \frac{dR_h}{dt}$ (.9)

and $\frac{dN_h}{dt} = \Pi_h + \Lambda_h - \delta_h I_h - \mu_h N_h$ (.10)

From equation (.10) if disease-induced death is removed then:

$$\frac{dN_h}{dt} \leq \Pi_h + \Lambda_h - \mu_h N_h; \text{ and hence, } \lim_{t\to\infty} N_h(t) \leq \frac{(\Pi_h + \Lambda_h)}{\mu_h}.$$

From the model equations (.5) to (.7) the total population size, N_v, at time *t* is given by:

$$N_v(t) = S_v(t) + E_v(t) + I_v(t) \quad (.11)$$

This then implies that: $\frac{dN_v}{dt} = \frac{dS_v}{dt} + \frac{dE_v}{dt} + \frac{dI_v}{dt}$ (.12)

$$\text{and } \frac{dN_v}{dt} \leq \Lambda_v - \mu_v N_v; \text{ hence, } \lim_{t\to\infty} N_v(t) \leq \frac{\Lambda_v}{\mu_v}.$$

Model fitting, validation and parameter estimation

A least-squares estimation method in Microsoft excel 2019 was used to fit the model system (.1) to (.7). Model calibration was done using the full dataset from 2015 – 2020. The dataset for December 2020 – February 2021 was used to assess and strengthen the model's predictive abilities. Parameter estimation and model simulation were carried out in Microsoft excel 2019.

Results

The disease-free equilibrium point was determined and the local stability of the system was described in terms of the threshold parameter, the basic reproduction number (R_0), which was obtained using the next generation matrix method.

Existence of the disease-free equilibrium (DFE) point

The DFE point are steady state solutions where there is no malaria in the human population, and no plasmodium parasites in mosquitoes. Thus the DFE point, given by $E_{0hv}(S_h, E_h, I_h, R_h; S_v, E_v, I_v)$ of the system of differential equations .1 to .7, is obtained by solving the equation

$$\frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0 \qquad (1.1)$$

The "infected" classes in the human or mosquito populations are either the exposed or infectious, that is E_h, I_h, R_h, E_v, I_v. So in the absence of disease, at the DFE point of the system .1 to .7, it implies $E_h = I_h = R_h = E_v = I_v = 0$; $S_h = \frac{\Pi_h + \Lambda_h}{\mu_h}$; and $S_v = \frac{\Lambda_v}{\mu_v}$.

Thus the DFE point $E_{0hv}(S_h, E_h, I_h, R_h; S_v, E_v, I_v)$ is given by

$$E_{0hv}\left(\frac{\Pi_h + \Lambda_h}{\mu_h}, 0, 0, 0; \frac{\Lambda_v}{\mu_v}, 0, 0\right).$$

As such, the positive feasible region of the system for the human population, equations .1 to .4 is given by

$$\left\{(S_h, E_h, I_h, R_h): S_h + E_h + I_h + R_h \leq \frac{\Pi_h + \Lambda_h}{\mu_h}, S_h > 0, E_h \geq 0, I_h \geq 0, R_h \geq 0\right\}.$$

The positive feasible region of the model equations .5 to .7 for the mosquito population is given by

$$\left\{(S_v, E_v, I_v): S_v + E_v + I_v \leq \frac{\Lambda_v}{\mu_v}, S_v > 0, E_v \geq 0, I_v \geq 0\right\}.$$

It follows therefore that the DFE point, $E_{0hv}\left(\frac{\Pi_h+\Lambda_h}{\mu_h}, 0, 0, 0; \frac{\Lambda_v}{\mu_v}, 0, 0\right)$, of the system of equations .1 to .7 is locally asymptotically stable if $R_{0hv} < 1$ and unstable if $R_{0hv} > 1$, where $R_{0hv} = \sqrt{R_{0h}R_{0v}}$, [23]. The square root is as a result of the needed two generations for an infected vector or host to reproduce itself [24]. R_{0hv} is the basic reproduction number of the system, while R_{0h} and R_{0v} are the basic reproduction numbers for human and mosquito populations, respectively.

The basic reproduction number, R_0

The basic reproduction number, R_0, is defined as the number of secondary infections that one infectious individual generates over the duration of his/ her entire infectious period, assuming that all the individuals in the population are susceptible.

When $R_0 < 1$, each infected individual produces, on average, less than one new infected individual and hence it is expected that the disease will die out. On the other hand, if $R_0 > 1$, each individual produces more than one new infected individual, so it would be expected that the disease will spread through the population. Therefore, the threshold condition for eliminating or eradicating the disease is to reduce the value of R_0 to less than one.

We introduce new variables in terms of proportion as follows:

$$a = \frac{\Pi_h}{N_h}, \quad b = \frac{\Lambda_h}{N_h}, \qquad s = \frac{S_h}{N_h}, \qquad e = \frac{E_h}{N_h}, \qquad i = \frac{I_h}{N_h}, \quad r = \frac{R_h}{N_h};$$

$$d = \frac{\Lambda_v}{N_v}, \qquad x = \frac{S_v}{N_v}, \qquad y = \frac{E_v}{N_v}, \quad z = \frac{I_v}{N_v}. \tag{1.2a}$$

where

$$s + e + i + r = 1; \;\; x + y + z = 1; \tag{1.2b}$$

$$s = (1 - e - i - r), \quad and \quad x = (1 - y - z);$$

$$S_h = sN_h \;\; and \;\; S_v = xN_v$$

Hence

$$S_h = (1 - e - i - r)N_h, \text{ and } S_v = (1 - y - z)N_v. \tag{1.2c}$$

Now from equations (1.2a), it can be seen that:

$$N_h \frac{ds}{dt} = \frac{dS_h}{dt} - s\frac{dN_h}{dt} \tag{1.3a}$$

$$N_h \frac{de}{dt} = \frac{dE_h}{dt} - e\frac{dN_h}{dt} \tag{1.3b}$$

$$N_h \frac{di}{dt} = \frac{dI_h}{dt} - i\frac{dN_h}{dt} \tag{1.3c}$$

$$N_h \frac{dr}{dt} = \frac{dR_h}{dt} - r\frac{dN_h}{dt} \tag{1.3d}$$

$$N_v \frac{dx}{dt} = \frac{dS_v}{dt} - x\frac{dN_v}{dt} \qquad (1.3e)$$

$$N_v \frac{dy}{dt} = \frac{dE_v}{dt} - y\frac{dN_v}{dt} \qquad (1.3f)$$

$$N_v \frac{dz}{dt} = \frac{dI_v}{dt} - z\frac{dN_v}{dt} \qquad (1.3g)$$

Using the relationships of equations (1.2) and (1.3), and re-writing the system equations (.1) to (.7) gives the following equations:

$$\frac{ds}{dt} = a + b + \rho_h r - \phi\beta_h sz - (a + b - \delta_h i)s \qquad (1.4)$$

$$\frac{de}{dt} = \phi\beta_h sz - (a + b + \alpha_h - \delta_h i)e \qquad (1.5)$$

$$\frac{di}{dt} = \alpha_h e - (a + b + \delta_h + \lambda_h - \delta_h i)i \qquad (1.6)$$

$$\frac{dr}{dt} = \lambda_h i - (a + b + \rho_h - \delta_h i)r \qquad (1.7)$$

$$\frac{dx}{dt} = d - \beta_v xi - c\beta_v xr - dx \qquad (1.8)$$

$$\frac{dy}{dt} = \beta_v xi + c\beta_v xr - (\gamma_v + d)y \qquad (1.9)$$

$$\frac{dz}{dt} = \gamma_v y - dz \qquad (1.10)$$

The infected classes of the system (.1) to (.7), and hence system (1.4) to (1.10), are now *e, i, r, y and z*. The next generation matrix method was used to determine the R_0 of the present model. The R_0 is taken to be the dominant Eigenvalue of the next generation matrix. This is given by the matrix FV^{-1}, where F represents the rate of appearance of new infections in the compartments and V represents the transfer of individuals in and out of compartments and E_0 is the disease-free equilibrium point. Thus

$$\mathcal{F}\begin{bmatrix} e \\ i \\ r \\ y \\ z \end{bmatrix} = \begin{bmatrix} \phi\beta_h sz \\ 0 \\ 0 \\ \beta_v xi + c\beta_v xr \\ 0 \end{bmatrix}$$

$$F(E_0) = \begin{bmatrix} 0 & 0 & 0 & 0 & \phi\beta_h \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_v & c\beta_v & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}.$$

And

$$\mathcal{V}\begin{bmatrix} e \\ i \\ r \\ y \\ z \end{bmatrix} = \begin{bmatrix} (a+b+\alpha_h-\delta_h i)e \\ -\alpha_h e + (a+b+\delta_h+\lambda_h-\delta_h i)i \\ -\lambda_h i + (a+b+\rho_h-\delta_h i)r \\ (d+\gamma_v)y \\ -\gamma_v y + dz \end{bmatrix}$$

$$V(E_{0)} = \begin{bmatrix} (a+b+\alpha_h) & 0 & 0 & 0 & 0 \\ -\alpha_h & (a+b+\delta_h+\lambda_h) & 0 & 0 & 0 \\ 0 & -\lambda_h & (a+b+\rho_h) & 0 & 0 \\ 0 & 0 & 0 & (d+\gamma_v) & 0 \\ 0 & 0 & 0 & -\gamma_v & d \end{bmatrix}$$

Let $a_{11} = (a + b + \alpha_h),\ a_{21} = -\alpha_h,\ a_{22} = (a + b + \delta_h + \lambda_h),\ a_{32} = -\lambda_h, a_{33} = (a + b + \rho_h),\ a_{44} = (d + \gamma_v);\ a_{54} = -\gamma_v$ and $a_{55} = d$. Then it implies:

$$|V| = a_{11}a_{22}a_{33}a_{44}a_{55}.$$

Let $\boldsymbol{V}$ be a vector V as a matrix of its co-factors, it then implies:

$$\boldsymbol{V} = \begin{bmatrix} a_{22}a_{33}a_{44}a_{55} & -a_{21}a_{33}a_{44}a_{55} & a_{21}a_{32}a_{44}a_{55} & 0 & 0 \\ 0 & a_{11}a_{33}a_{44}a_{55} & -a_{11}a_{32}a_{44}a_{55} & 0 & 0 \\ 0 & 0 & a_{11}a_{22}a_{44}a_{55} & 0 & 0 \\ 0 & 0 & 0 & 0 & -a_{11}a_{22}a_{33}a_{54} \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}, \text{ then}$$

$$\boldsymbol{V^T} = \begin{bmatrix} a_{22}a_{33}a_{44}a_{55} & 0 & 0 & 0 & 0 \\ -a_{21}a_{33}a_{44}a_{55} & a_{11}a_{33}a_{44}a_{55} & 0 & 0 & 0 \\ a_{21}a_{32}a_{44}a_{55} & -a_{11}a_{32}a_{44}a_{55} & a_{11}a_{22}a_{44}a_{55} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -a_{11}a_{22}a_{33}a_{54} & 0 \end{bmatrix}$$

$$V^{-1} = \frac{1}{|V|}\boldsymbol{V^T} \qquad (1.11)$$

$$= \begin{bmatrix} \frac{1}{a_{11}} & 0 & 0 & 0 & 0 \\ \frac{-a_{21}}{a_{11}a_{22}} & \frac{1}{a_{22}} & 0 & 0 & 0 \\ \frac{a_{21}a_{32}}{a_{11}a_{22}a_{33}} & \frac{-a_{32}}{a_{22}a_{33}} & \frac{1}{a_{33}} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{-a_{54}}{a_{44}a_{55}} & 0 \end{bmatrix}$$

$$FV^{-1} = \begin{bmatrix} 0 & 0 & 0 & \frac{-\phi\beta_h a_{54}}{a_{44}a_{55}} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ \left(\frac{-\beta_v a_{21}}{a_{11}a_{22}} + \frac{c\beta_v a_{21}a_{32}}{a_{11}a_{22}a_{33}}\right) & \left(\frac{\beta_v}{a_{22}} - \frac{c\beta_v a_{32}}{a_{22}a_{33}}\right) & \frac{c\beta_v}{a_{33}} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

Therefore, the basic reproduction number (R_{0hv}) is thus given by:

$$R_{0hv} = \sqrt{R_{0h}R_{0v}}$$

Therefore:

$$R_{0hv} = \sqrt{\left\{\left(\left[\frac{\alpha_h\beta_v}{(a+b+\alpha_h)(a+b+\delta_h+\lambda_h}\right] + \left[\frac{c\beta_h\alpha_h\lambda_h}{(a+b+\alpha_h)(a+b+\delta_h+\lambda_h)(a+b+\rho_h)}\right]\right).\left(\frac{\emptyset\beta_h\gamma_v}{d(d+\gamma_v)}\right)\right\}}$$

$$R_{0hv} = \sqrt{\frac{\emptyset\beta_h\gamma_v[\alpha_h\beta_v(a+b+\rho_h) + c\beta_v\alpha_h\lambda_h]}{d(d+\gamma_v)(a+b+\alpha_h)(a+b+\delta_h+\lambda_h)(a+b+\rho_h)}}$$

$$= \sqrt{\frac{\emptyset\beta_h\beta_v\gamma_v\alpha_h[a+b+\rho_h+c\lambda_h]}{d(d+\gamma_v)(a+b+\alpha_h)(a+b+\delta_h+\lambda_h)(a+b+\rho_h)}}$$

= 0.096906348 (from Table 4); $R_{0hv} < 1$.

Malaria transmission is here depicted as resulting from transmission of the plasmodia parasites from infected humans to susceptible mosquitoes and from infected mosquitoes to susceptible humans. The R_{ohv} here is related to the transmission of disease by infectious humans in the I compartment, and/or partially immune recovered in the R compartment to susceptible mosquitoes. Susceptible humans are infected following infectious bites by infected mosquitoes. Thus, near the DFE (E_{0hv}) point, each infected human is likely to produce $\frac{\beta_v\gamma_v}{d(d+\gamma_v)}$ new infected mosquitoes, while each infected mosquito is likely to produce $\frac{\emptyset\beta_h\alpha_h[a+b+\rho_h+c\lambda_h]}{(a+b+\alpha_h)(a+b+\delta_h+\lambda_h)(a+b+\rho_h)}$ new infected humans during their infectious period.

***Stability analysis of the DFE (E_{0hv})* point**

The DFE (E_{0hv}) point is locally asymptotically stable if $R_{0hv} < 1$ and unstable if otherwise. If the $R_{0hv} < 1$, then the solutions of the equation $J|E_{0hv} - \lambda I| = 0$ are all negative.

Theorem: Considering the disease transmission model given by equations (.1) to (.7) re-written as equations (1.4) to (1.10). If E_{0hv} is the DFE point of the model, then E_{0hv} is locally asymptotically stable if the eigenvalues of the matrix (1.12) are all negative.

Proof: The Jacobian of the system (1.4) to (1.10) is obtained as

$$J(s,e,i,r;x,y,z) = \begin{bmatrix} \boldsymbol{A} & 0 & \delta_h s & \rho_h & 0 & 0 & -\emptyset\beta_h s \\ \emptyset\beta_h z & \boldsymbol{B} & \delta_h e & 0 & 0 & 0 & \emptyset\beta_h s \\ 0 & \alpha_h & \boldsymbol{C} & 0 & 0 & 0 & 0 \\ 0 & 0 & \lambda_h & \boldsymbol{D} & 0 & 0 & 0 \\ 0 & 0 & -\beta_v x & -c\beta_v x & -(\beta_v i + c\beta_v r) - d & 0 & 0 \\ 0 & 0 & \beta_v x & c\beta_v x & \beta_v i + c\beta_v r & -(\gamma_v + d) & 0 \\ 0 & 0 & 0 & 0 & 0 & \gamma_v & -d \end{bmatrix}$$

Where

A $= -\emptyset\beta_h z - (a + b - \delta_h i)$;

B $= -(\alpha_h + a + b - \delta_h i)$;

C $= -(a + b - \delta_h i + \delta_h + \lambda_h)$;

D $= -(\rho_h + a + b - \delta_h i)$.

When the system is evaluated at the DFE point, E_{0hv}, it becomes

$$J(E_{0hv}) = \begin{bmatrix} -(a+b) & 0 & \delta_h & \rho_h & 0 & 0 & -\emptyset\beta_h \\ 0 & -(\alpha_h + a + b) & 0 & 0 & 0 & 0 & \emptyset\beta_h \\ 0 & \alpha_h & -(a+b+\delta_h+\lambda_h) & 0 & 0 & 0 & 0 \\ 0 & 0 & \lambda_h & -(\rho_h + a + b) & 0 & 0 & 0 \\ 0 & 0 & -\beta_v & -c\beta_v & -d & 0 & 0 \\ 0 & 0 & \beta_v & c\beta_v & 0 & -(\gamma_v + d) & 0 \\ 0 & 0 & 0 & 0 & 0 & \gamma_v & -d \end{bmatrix}$$

Therefore, solving the matrix equation

$$J|E_{0hv} - \lambda I| = 0 \quad \text{gives} \qquad (1.12)$$

$$\begin{bmatrix} -(a+b)-\lambda & 0 & 0 & \rho_h & 0 & 0 & -\emptyset\beta_h \\ 0 & \boldsymbol{X} & 0 & 0 & 0 & 0 & \emptyset\beta_h \\ 0 & \alpha_h & \boldsymbol{Y} & 0 & 0 & 0 & 0 \\ 0 & 0 & \lambda_h & \boldsymbol{Z} & 0 & 0 & 0 \\ 0 & 0 & -\beta_v & -c\beta_v & -d-\lambda & 0 & 0 \\ 0 & 0 & \beta_v & c\beta_v & 0 & -(\gamma_v + d) - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & \gamma_v & -d-\lambda \end{bmatrix} = 0 \qquad (1.13).$$

Where

X $= -(\alpha_h + a + b) - \lambda$

Y $= -(a + b + \delta_h + \lambda_h) - \lambda$

Z $= -(\rho_h + a + b) - \lambda.$

The above (1.13) then reduces to

$$(-(a+b)-\lambda)\{[-(\alpha_h+a+b)-\lambda][-(a+b+\delta_h+\lambda_h)-\lambda][-(\rho_h+a+b)-\lambda](-d-\lambda)[-(\gamma_v+d)-\lambda](-d-\lambda)\}-\emptyset\beta_h\beta_v\gamma_v\alpha_h(-(a+b)-\lambda)(-c\lambda_h - (a+b)-\lambda)\}=0.$$

It then implies that

$$(-(a+b)-\lambda)(-d-\lambda)\{[-(\alpha_h+a+b)-\lambda][-(a+b+\delta_h+\lambda_h)-\lambda][-(\rho_h+a+b)-\lambda][-(\gamma_v+d)-\lambda]\}-\emptyset\beta_h\beta_v\gamma_v\alpha_h(-c\lambda_h-(\rho_h+a+b)-\lambda)\}=0.$$

Thus the solutions to equation above are $\lambda_1=-(a+b);\ \lambda_2=-(\alpha_h+a+b);\ \lambda_3=-(a+b+\delta_h+\lambda_h);\ \lambda_4=-(\rho_h+a+b);\ \lambda_5=-d;\ \lambda_6=-(\gamma_v+d), \lambda_7=-\big(c\lambda_h+(\rho_h+a+b)\big)$

Since the solutions of the model are all negative it can be concluded that the E_{0hv} is locally asymptotically stable, and hence, the $R_{0hv}<1$.

Summary statistics from Masvingo district malaria data

The following table, Table 2, shows the descriptive statistics of monthly confirmed Rapid Diagnostic Test (RDT) positive malaria cases disaggregated by age and sex from 2015 to 2020.

From Table 2, it can be seen that the mean number of confirmed positive malaria cases increases with age.

Table 3 presents summary statistics of the malaria incidence disaggregated by sex and year of occurrence. It can be seen that males generally contribute more malaria cases compared to the female population.

Numerical simulation and projection

Microsoft Excel (2019) was used for parameter estimation and to study the dynamical behavior of the system of equations (1.4) to (1.10). To simplify these model equations, Euler's method was used. It states that if

$$\frac{dA}{dt}=-kA+hB^2, and\ LHS=\frac{A_{i+1}-A_i}{\Delta_t}$$

$$\text{then}, A_{i+1} = (-kA + hB^2) * \Delta_t + A_i.$$

In the context of malaria transmission in Masvingo district, the parameter values chosen are shown in table 4, and the following initial conditions were selected: $S_h = 299101;\ E_h = 59821;\ I_h = 26;\ \ R_h = 0$; and the best model fit, determined by the square of the Pearson's product moment correlation coefficient ($R^2 = 0.8923$) passing through the data provided is shown in Figure 2. RDNS- positive cases (infected) refers to the individuals who tested malaria positive and notified according to the Rapid Disease Notification System (RDNS). Expected malaria cases were obtained after applying the model to the existing disease data (cases), to see how best it fits the data. The 2-period moving average was obtained by finding the average of two consecutive values which seeks to smooth-out month-to-month fluctuations.

The trend in the incidence of malaria over time is shown. Although there is a general decline in the number of malaria cases, the transmission dynamics is highly fluctuating (Figure 2).

Assuming that there won't be any drug resistance or insecticide resistance, malaria cases are likely to drop to below one (1) cases per month by the 144th month (December 2026) (Figure 3). This shows that there is great potential for malaria elimination by the year 2030.

Discussion

An ordinary differential equation-based compartmental model for the transmission of falciparum malaria was formulated for both human and mosquito populations. The basic assumptions underlying the model formulation were stated. The human population was modeled using the SEIR model while the mosquito population was modeled using the SEI model. In formulating the model, humans recovered with partial immunity which waned over time and thus became susceptible again. However, in their recovered state, these humans habour the infectious parasite and thus can infect susceptible mosquitoes but at a lower rate compared to the rate by infectious humans. Model analysis was also done. The existence of a disease free equilibrium (DFE) point was ascertained and was found to be locally asymptotically stable.

The basic reproduction number, which is a threshold parameter, was derived and it was shown to be a square-root of the product of the vector reproduction number and the human reproduction number [20], [21]. The obtained reproduction number (R_{0hv}) shows that in the scenario represented by the model, the disease will ultimately die out. This would mean that fewer than one infection, in both mosquito and human populations, will be generated from a single infected individual. This subsequently reduces incidence over time. The recognition of reducing R_{0hv} to less than 1 has prevention and control connotations: most modern epidemiological practices aim to reduce the reproduction number as the primary focus of infectious disease management. This approach has implications and is critical when aiming for malaria elimination by 2030 [3]. As reported elsewhere, there is great potential for malaria as the disease has been eliminated from about 79 countries between 1979 and 2010 [18]. Disease forecast has shown that beyond the 144th month, February 2027, (figure 3) the number of infected individuals will decline to below 1. This way, the result is helpful in predicting the disease trend in the district which assist in evaluating efforts towards disruption of local transmission, especially so as the district targets zero local malaria transmission by 2030. As has been reported [18]., malaria was eliminated from about 79 countries between 1979 and 2010, and according to the report [23], one district in Zimbabwe had been verified and declared to have eliminated local malaria transmission. However, since the data used in this study was highly fluctuating, possibilities of outbreaks of malaria cannot be ruled out. Consequently, as the district approaches zero local cases caution should be taken to guard against the rebound of the disease.

According to research [13] at low incidence, mosquito bites will also be low, and since immunity is conferred through continued exposure to mosquito bites [12], individual immunity will wane, and with an introduction of infected mosquito(es) the disease can bounce back with bouts of cases being experienced. In this study, only a single intervention (LLINs) was modelled as the dominant strategy. However, some other interventions such as case management, behavior change and communication strategies and foci management are also relied on, though they were not modelled in this study. As such further research may be needed which models may incorporate such factors in a bid to formulate a more robust model. Again, in the study aggregated data was used. The age and sex of an individual affects exposure to mosquito bites and subsequent risk of disease. In this study, the mean number of confirmed positive malaria cases were seen to increase with age. This is consistent with observations made by some authors [24] , [25]that as one grows older he or she presents a greater surface area available for mosquito bites. As a result, adults seem to have an increased exposure to mosquito bites and risk of suffering from malaria. Again, males contributed more malaria cases compared to their female counterparts. This may be due to the fact that the male population spend greater part of their time outdoors where they get exposed to mosquito bites. However, further modelling studies utilizing disaggregated data by age or sex are suggested. These may yield more reliable evidence as the modelling will take into consideration the variation in disease risk between population categories. While this study provides insights into the district's malaria transmission dynamics, contextual variations such as socio-economic and cultural factors across districts may affect generalizability of these findings. Future research, however, may consider incorporating diverse districts to enhance robustness and generalizability of the conclusions.

Conclusion

This study developed a compartmental model for the transmission of falciparum malaria using ordinary differential equations for both human and mosquito populations. The fundamental presumptions that guided the creation of the model were outlined. The combination impacts of recruitment rates, migration, recovery with temporary immunity and subsequent loss thereof, and infectiousness of the recovered humans – albeit at a lower rate than infectious humans – were demonstrated by the formulation and analysis of the falciparum malaria transmission model.
The DFE point was shown to exist, and to be locally asymptotically stable when $R_{0hv} < 1$. When $R_{0hv} < 1$ the disease will eventually die off.

With a transmission rate of less than one local cases, the model's numerical simulation demonstrated that, despite the highly variable nature of the transmission dynamics in Masvingo district, the incidence is generally declining. This declining trajectory signifies that the district is on track towards the achievement of WHO 2030 local falciparum malaria elimination target.
Sustaining and building on this trajectory is paramount and requires a multi-faceted approach as well as continued commitment by:

- Promoting behavior change communication to encourage continued widespread distribution and proper usage of long-lasting insecticidal nets (LLINs)
- Ensuring availability and accessibility of rapid diagnostic tests (RDTs) and effective anti-malarial treatment
- Formulating strategies to maintain transmission at low rates thereby preventing resurgence as well as planning for effects of waning immunity at low transmission rates and challenges associated with malaria drug resistance
- Fostering collaborations between research institutions and other stakeholders for better data collection and utilization

What is already known about the topic

- The disease incidence rate is at 4 cases per 1000 PAR
- The district is targeted for local falciparum malaria elimination by the year 2030
- The district is currently using long-lasting insecticidal nets (LLINs), intermittent preventive treatment for pregnant women and infants, case detection, investigation and management

What this study adds

- Deriving the reproduction number for this model provides a quantitative parameter to understand the trajectory for malaria transmission dynamics in the district, and having a reproduction number less than one shows a potential for achieving zero local transmission
- By modelling the human-mosquito interplay underscores the importance of targeting both human and mosquito populations in malaria control as the district strives to achieve zero local malaria transmission
- The model's prediction that cases could drop to below one case per month by December 2026, given no drug insecticide resistance, gives hope for elimination efforts by providing data-driven insights for continued guided malaria control program planning
- This study also provides a foundation for future research. Any future research can build on this study by incorporating additional factors, such as disaggregated data, to enhance robustness of the model.

Competing Interest

The Authors declare no competing interest

Authors´ contributions

Taremba Chirigo conceptualized the study; Taremba Chirigo and Esther Chigidi designed the study; Herbert Tafadzwa Kazonda searched for literature; Taremba Chirigo and Esther Chigidi developed data analysis techniques and analysed the data; Taremba Chirigo drafted the manuscript; Taremba Chirigo, Esther Chigidi and Hebert Tafadzwa Kazonda edited the manuscript. All the authors read and approved the final manuscript.

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Table 1: List of parameters for the malaria model and their definitions	
Parameter	Description
Λ_h	Recruitment rate of humans through natural birth
Λ_v	Recruitment rate of mosquitoes
Π_h	Susceptible immigrants into Masvingo district
ϕ	Rate constant due to non-use of preventive measures such as LLINs
β_h	Infection rate of susceptible humans, given by a product of *mosquito biting rate* and *probability of transmission of infection from an infectious bite to a susceptible human.*
β_v / $c\beta_v$;	Infection rate of susceptible mosquitoes, given by a product of *mosquito biting rate* and *probability of infection transmission from an infectious/ recovered human to a susceptible mosquito.*
c; $0 < c < 1$	Proportionality constant signifying a lower probability of transmission from a recovered compared to an infected human
α_h	Progression rate of humans from the exposed state to being infectious
λ_h	Rate of recovery with temporary immunity
ρ_h	Rate of loss of immunity by recovered humans
δ_h	Disease-induced death rate for humans
γ_v	Progression rate of mosquitoes from exposed to infectious state
μ_h	Natural death rate of humans
μ_v	Natural death rate of mosquitoes

Table 2: Descriptive statistics of monthly confirmed (Rapid Diagnostic Test positive) malaria cases disaggregated by age, Masvingo district 2015 -2020

Variable	Number of months/ Observation	Mean number of cases	Standard deviation	Minimum number of cases	Maximum number of cases
Malaria Confirmed Cases in Males under 5 Years	72	4.125	1.7198	0	31
Malaria Confirmed Cases in Females under 5 Years	72	3.13889	4.237012	0	19
Malaria Confirmed Cases, Males, 5+ Years	72	38.76389	51.78898	2	255
Malaria Confirmed Cases, Females 5+ Years	72	32.54167	45.07738	0	215
All Males	72	42.88889	57.56406	0	255
All Females	72	35.68056	48.92933	0	215

Table 3: Descriptive statistics for malaria occurrence in Masvingo district disaggregated by sex, 2015 - 2020

Period/ Year	Sex	Mean of malaria cases	Standard deviation, *sd*	Standard error, *se* (mean)	Sum of cases	Minimum number of cases	Maximum number of cases
2015	**Male**	30.667	31.008	8.951	368	4	111
	Female	27.833	26.405	7.623	334	2	93
2016	**Male**	20.583	8.785	2.536	247	7	32
	Female	16.5	8.152	2.353	198	2	29
2017	**Male**	67.5	56.179	16.218	810	19	226
	Female	58.917	54.176	15.639	707	16	215
2018	**Male**	52.75	72.644	20.97	633	5	2128
	Female	42.667	62.261	18.159	512	1	184
2019	**Male**	42.917	72.261	20.86	515	2	255
	Female	34.083	57.80	16.684	409	0	201
2020	**Male**	42.917	72.261	20.86	515	2	255
	Female	34.083	57.80	16.684	409	0	201
Total	**Male**	42.889	57.564	6.784	3088	2	255
	Female	35.681	48.929	5.766	2569	0	215

Table 4: Model parameter values		
Parameter/ Symbol	**Value/ Estimate**	**Source**
a	0.011651316	(22)
b	0.001791748	(22)
d	0.1429	(20)
ϕ	0.2	Estimated
β_h	*0.02*	(20)
β_v	*0.833*	(20)
$c; 0 < c < 1$	*0.1*	(20)
α_h	2.93102E-05	Estimated
λ_h	0.156699629	Estimated
ρ_h	0.022	Estimated
δ_h	0.000011	Estimated
γ_v	0.1	(20)
μ_h	0.000146	(22)
μ_v	0.1429	(20)

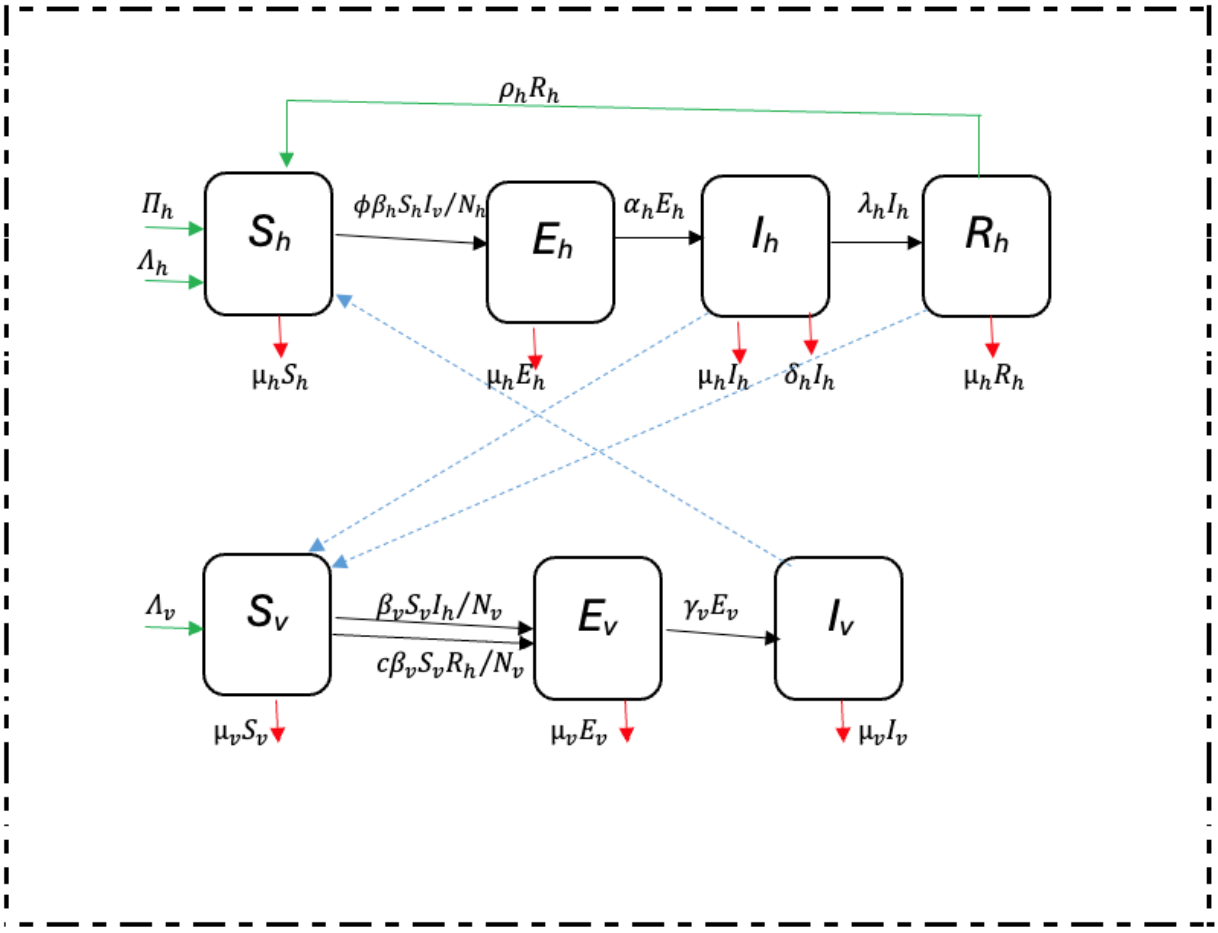

Figure 1: Interaction of the different classes of human and mosquito populations. A modified Ngwa and Shu model.

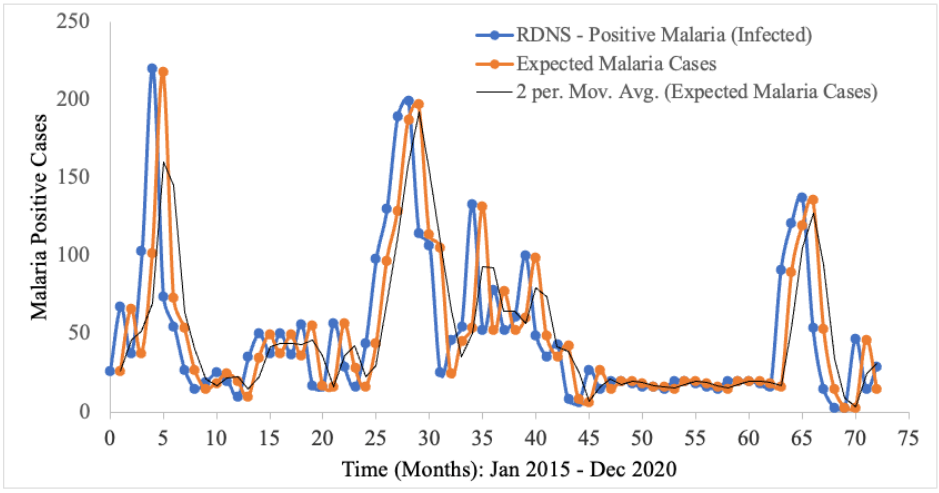

Figure 2: Model fitting to Masvingo district malaria data

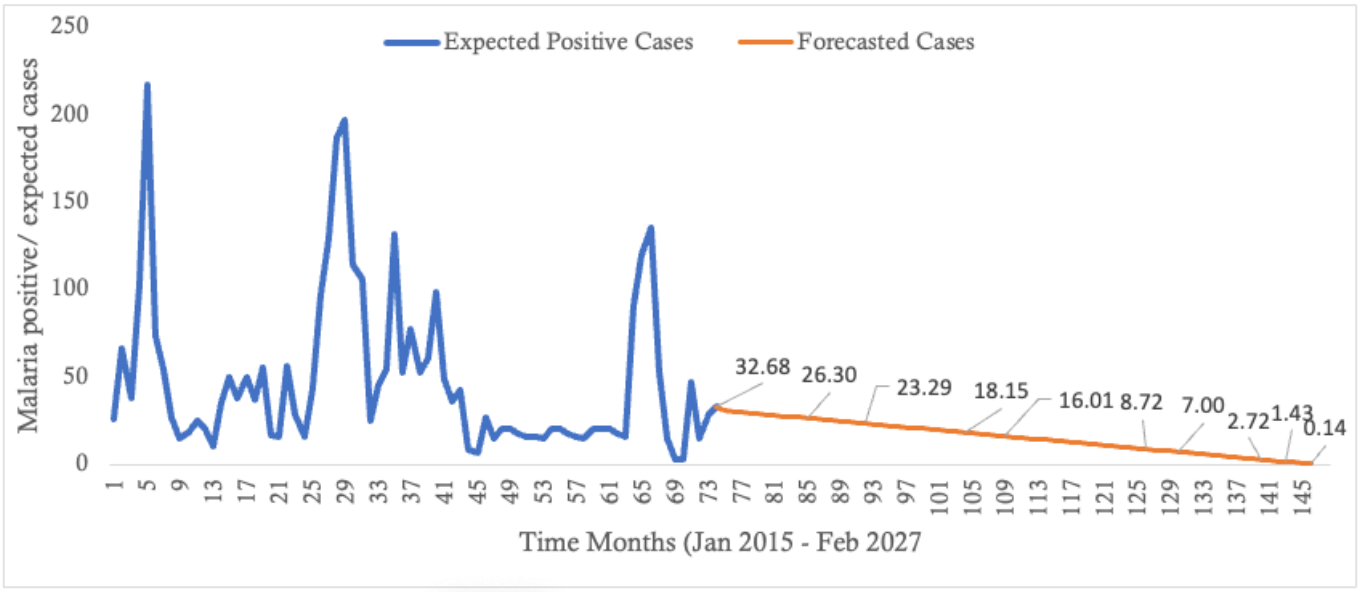

Figure 3: Trendline of Malaria Cases in Masvingo district with a forecast as from the 74th month