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Modeling the impacts of income inequality on malaria transmission dynamics

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ABSTRACT
Recently, a general agreement has come out to support the fact that improved economic conditions for households and individuals generally leads to better health conditions. Due to income inequality in society, some households and individuals cannot afford quality treatment against malaria. We examine how income inequality affects malaria transmission dynamics by dividing the total human population broadly into; low income, middle income, and high-income earners. We then formulated a system of deterministic differential equations based on income inequalities to study malaria transmission dynamics. Impacts of income-inequality are investigated through the incorporation of the Gini index into the model equations. We find that the diseasefree equilibrium point of the model always exists. Using the center manifold theorem, we find a condition under which backward bifurcation will occur in the model. We also find that even without a disease-induced death rate, backward bifurcation may exist. Numerical simulations conducted show that widening income inequality will increase malaria cases and the number of people being hospitalized.

Keywords: *Bifurcation Gini index Income Income inequality*.

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I. INTRODUCTION

Malaria is widely considered as one of the most severe global public health problems worldwide, particularly among tropical regions of the world. In Africa, Nigeria is reported to have the greatest number of malaria cases, [19]. The disease is a major public health concern in sub-Saharan Africa as it is a leading cause of disability and death, especially among children. It was reported that in 2016 alone, 216 million people world over suffered from malaria with over 400,000 deaths,[50] . Since the year 2000, substantial progress has been made in curbing the menace of malaria. From the report in [59] , it was noted that between 2000 and 2015 , malaria case incidence and mortality rates were decreased by 41% and 62% respectively and endemic regions were decreased from 108 countries and territories in the year 2000 to 91 in 2016. These successes are generally linked to early diagnoses, drug-therapies, better health infrastructure, use of insecticide-treated bed-nets (ITNs), Long-lasting insecticide nets (LLINS), intermittent preventive treatment especially for pregnant women during anti-natal, and indoor residual spraying [19, 11, 33, 38, 44, 61]. Despite the remarkable progress, malaria remains the most deadly vector-borne disease in the world. Malaria has a ravaging touch on public health and socio-economic conditions of the people by imposing a heavy economic burden on individuals, households, and the entire economy [18, 23, 59].

There are many reports in the literature suggesting that wealth and income distributions have significant impacts on malaria transmission and health in general. For example, the report in [52] assert that income inequality has negative impacts on education and health. According to Michael Marmot reported in [43], income is related to health through the gross national product of countries, the income of individuals, and the income inequalities that exist among rich nations and geographic areas. Similarly, one of the findings in the empirical work reported in [19] is that the prevalence of malaria was associated significantly with having a low household family income, not using ITNs, and having no toilets in the house among other factors. In that report, household family income was divided into two based on monthly income, where the low income was considered to constitute a significant risk group. Otto Lenhart reported in [41] found that families with higher income may have better access to health care, whereas people with lower income are likely to be faced with more stressful situations that are

detrimental to health. Perhaps this finding might be explained further by the report in [52], where it was shown that about 26% of the National monthly minimum wage in Nigeria is required to cover the cost of uncomplicated malaria treatment per patient per malaria episode in some private hospitals in Ibadan, Nigeria. Similarly, one of the conclusions of the report in [7] is that the cost of malaria treatment for under-five children in some parts of Ghana is considerably high in comparison to the poverty level of the area. The implication of this is that lack of fund makes malaria patients resort to self-treatment or no treatment at all, see [7]. Other reports on the negative effects of low income on malaria treatment in some African countries such as Ethiopia, Mozambique, Tanzania, Togo, and Nigeria can be found in [17, 4, 35, 45, 55]. In fact, the connection between, malaria, poverty and income inequality is well documented in [29, 53]. The distribution of wealth in most countries for which there is reliable data is strikingly uneven. According to the report in [59], the world is more unequal today than any other time since World War II and that the richest 1% of the world population owns about 40% of the world's assets, while the bottom half owns no more than 1%. As a result of income-inequality countries and communities of the world are divided into different groups such as low income, lower middle income, upper middle income, and the high-income groups, [36, 57]. The division is mainly based on Gini index which is a summary statistic with value in the range $[0,1]$ that measures how fairly income is distributed in a population, [24, 37]. Definition of Gini index is based on the Lorenz curve which plots the percentage, $L(x)$ of the total income of a population that is cumulatively earned by the bottom $x\%$ of the population. The more equal the distribution of income, the smaller the value of the Gini index and vice-versa.

The global targets of WHO and the global malaria community is a world free of malaria by 2030. As part of the contribution by researchers for the attainment of these targets, mathematical models of malaria transmissions are developed by several authors to gain insight into the dynamics of the disease. In these models, several characteristic features of malaria disease such as clinical immunity, malaria-strains, and protections against the disease are investigated and analyzed mathematically [6, 27, 39, 49]. Other features of malaria disease considered by modelers are personal/household protection against the disease through the use of ITNs and LLINs. For example, using global uncertainty and sensitivity analysis, Bala and Gimba reported in [6] concluded that malaria can be controlled through the combination of 95% drug treatment of malaria cases, and 95% ITNs coverage with 95% efficacy. Despite the significant achievement recorded on the use of ITNs in mitigating the scourge of malaria, certain problems associated with it are reported in the literature. Firstly, Briget and Koella, [40] reported that the use of ITNs is like a double-edged sword because it diverts infectious mosquitoes to non-users, and hence increasing their risk. Secondly, the report in [26] hypothesis that high use of ITNs may cause mosquitoes to adapt and change their biting to day time through evolutionary behavior. Thirdly, the requirements of regular re-treatment to preserve ITNs efficacy impose an additional cost on low-income earners, for this reason, LLINs, recommended by WHO and Roll Back Malaria partners is generally prefer as a cost-effective and sustainable method for protection against malaria [16]. From the report in [44], the authors concluded that using LLINs with over 90% coverage will lead to effective malaria control. However, reaching this coverage level can be quite challenging. From the report in [3], there have been studies in Tanzania, Madagascar, and India, where low demand for LLINs among mostly low-income households was found. The report further asserts that there exists a high demand for LLINs among the middle-income class in Ghana. This suggests that income difference will play a significant role in achieving a high level of LLINs coverage unless a strategy for subsidizing for the poor/low-income group is put in place, see [3, 32].

In the models reported in [1, 12], the authors incorporated a parameter in the mosquito biting rate function to model the impacts of ITNs usage. In the model reported in [46], the biting rate function not only contains ITNs coverage but also contains its efficacy and replacement time. Recent studies have shown that the concept of relying on malaria transmission through mosquito biting rates alone is flawed, [2, 14]. In these studies, it was shown that salivary gland sporozoite load strongly correlates with malaria infection probability. Thus, it is imperative to sporozoite load into account when modeling the malarial force of infection. As reported by many researchers, one of the characteristic features of malaria disease that makes control effort difficult is the phenomenon of backward bifurcation which has been observed in many malaria models. Some of these studies can be found in [10, 28, 30, 31, 39, 47]. Under this circumstance, the necessity for the basic reproduction number to be less than one for the disease to be controlled is no longer holds. The occurrences of backward bifurcation are typically linked to; disease-induced death rates and choice of incidence functions [25, 34]. Apart from dynamical models, there are health economic models of infectious diseases that have employed static conditions to study disease effects, see for instance [7, 15, 48, 51, 53, 58]. This type of approach may be

acceptable for malaria control where disease burden and transmission is constant. However, this is not the malaria case especially in the endemic regions and also for the fact that malaria transmission intensity is highly affected by seasonality [5, 8, 20, 42].

Widening income inequality is the defining challenge of our time. In many countries, the gap between the rich and poor is at its highest level in decades. Inequities have negative impacts on access to education, and health care. To make matters worse, many of the people in desperate need of malaria treatment simply cannot afford it. Not surprisingly then, the extent of inequality, its drivers, have become one of the topics of frequent discussion by policymakers and researchers alike. Against this background, we wish to combine dynamic mathematical modeling and some basic concepts in economics to formulate a model to investigate how income inequality affects malaria transmission and to find out whether there are other factors apart from disease-induced death rate that can bring about the phenomenon of backward bifurcation. To the best of our knowledge, this is the first time in literature where income inequality is incorporated into a dynamic model of malaria transmission to study its effects.

II. MODEL FORMULATION

We formulate a basic model to study the effects of income inequality on the malaria transmission dynamics by considering the human and mosquito populations. The human population is divided into 3 main groups; the low income, middle income, and high income earners. The low income group is made up of susceptible, exposed, infected, treated and recovered denoted by S_1, E_1, I_1, T_1 and R_1 respectively. Similarly, the middle income group is made up of susceptible, exposed, infected, treated and recovered denoted by S_2, E_2, I_2, T_2 and R_2 respectively. Also, the high income earners is made up of susceptible, exposed, infected treated and recovered individuals denoted by S_3, E_3, I_3, T_3 and R_3 respectively. The total human population is denoted by $N_h = \sum_{i=1}^{3} (S_i + E_i + I_i + T_i + R_i)$. All recruitment of new individuals are assumed to join one of the susceptible compartments at a rate Λ_i, for humans and Λ_4 for mosquitoes, unless otherwise stated, the subscript i is assumed to vary from 1 to 3 . The reason for chosen different recruitment rates for each susceptible class is in line with the report in [54], which states that individuals with improved economic conditions have lower birth rates. All human individuals can exit their current compartment due natural death, which can occur at a rate μ_h. We assumed that susceptible humans with low income can move to susceptible class with middle income through interaction with susceptible individuals with high income at a rate proportional to linearly decreasing function of the inequality-level measured by the Gini index g given by $\alpha_{13} = \alpha_1(1-g), \alpha_1$ is constant. We also assumed that individuals from susceptible class with middle income can move to the susceptible class with low income due to lose of jobs/income that might happen due to some circumstances such as the emergence of Covid-19, see for instance [22]. We modeled this transition as an increasing function of the Gini index given by $\alpha_{22} = \alpha_2 g, \alpha_2$ is constant. During economic growth developing countries may encounter difficulties in their transition from the middle income group to the high income group and will remain in the middle income group for several years, [21]. For this reason and also to reduce the complexity of the model equations, we assume that there is no transition from the middle income group to the high income group. Susceptible humans can be infected through the bite of infectious mosquito at a rate $\lambda_i(t)$ after which they move to the respective exposed compartments. The rate $\lambda_i(t)$ is the force of infection and it assumed to depend on the average number of mosquito bites ε, effect of personal protection against malaria, ℓ_i, the number of sporozoite per mosquito n_s, the average number of mosquito per human host ρ, as well as the transmission probability from infectious mosquito to human given that there is contact σ_h. We model the force of infection by

$$\lambda_i = \frac{b_i V_m(t)}{N_h(t)}$$

where $b_i = \varepsilon n_s \sigma_h \ell_i \rho$. The effect of personal protection is incorporated into the force of infection using

$$\ell_i = \zeta_{\max}(1 - \nu_i z_i) + \nu_i \zeta_{\min} z_i \qquad (2.0)$$

where $0 \le \nu_i, z_i \le 1$ represents the proportion of ITNs coverage and efficacy of ITNS respectively, $\zeta_{\min}, \zeta_{\max}$ represents minimum and maximum transmission rates respectively. We further assumed that $\nu_1 \le \nu_2 \le \nu_3$ and $z_1 \le z_2 \le z_3$ to account for the fact the higher the income, the higher the possibility of purchasing items required to reduce contacts with mosquitoes. The implication of this assumption is that $\ell_3 \le \ell_2 \le \ell_1$ and $\lambda_1 > \lambda_2 > \lambda_3$. The personal protection formula given in equation (2.0) is slightly different from the one used in [1] due to incorporation of ITNs efficacy, z_i is incorporated. Strictly speaking, bed-nets are generally used indoors and at certain times of the night, however early and outdoor biting by mosquitos have been reported in literature, see for instance [9, 56], hence, we follow the assumption made in [1] that even if the entire host population used fully efficient bed-nets $(\nu_i = 1, z_i = 1)$, the transmission can only be reduced to a minimum

value $\zeta_{\min} > 0$. Likewise, if nobody uses bed-nets ($\nu_i = 0$), transmission will be at its maximum level.

We assume that exposed humans who survives the latent period becomes infectious at the γ_i and move to the corresponding infectious compartment. Members of the infectious classes are either treated at a rate κ_i and enter the respective treatment class of treated infectious, or they may recover at a rate β_i without medication and enter the corresponding class of recovered humans R_i, or die from the disease at a rate δ_i. Members of the treatment compartments can recover or die from the disease at the rates $\eta_i\beta_i, \delta_i\theta_i$ respectively, where $0 < \theta_i \leq 1$, represents modification parameter to account for reduced death rate in the presence of the disease, $\eta_i \geq 1$, η_i measures the potency of the drug in reducing the disease-induced death of infectious humans, [13]. We assume that humans with high-income can afford high-quality drugs which might be expensive. Hence, the difference in the value of this parameter. Individuals in the recovered compartments can lose immunity and join the respective susceptible class at a rate ϕ_i. We assign weights (u_i) to each of the three income groups and using these weights we calculate $\eta_i, \theta_i, \kappa_i$, and ν_i using the Lorenz curve reported in [37],

$$L(u_i) = u_i^{\frac{2g}{1-g}}\left(1 - (1-u_i)^{\frac{1-g}{1+g}}\right). \quad (2.1)$$

In fact equation (2.1) comprises of the product of two Lorenz curves

$$L_1(u_i) = u_i^{\frac{2g}{1-g}}, \quad (2.2)$$

$$L_2(u_i) = \left(1 - (1-u_i)^{\frac{1-g}{1+g}}\right). \quad (2.3)$$

We assumed that $\eta_i = 1 + 2L(u_i), \theta_i = 1 + 2L_2(u_i), \kappa_i = L(u_i), \nu_i = \kappa_i$

The mosquito population is divided into two compartments, susceptible and infectious, which are denoted by S_m, V_m, respectively, with a total population given by $N_m = S_m + V_m$. We assumed that susceptible mosquitoes are recruited by birth at a fixed rate Λ_4, and this population is decreased by infection, following effective contacts with infected humans, at a rate $\lambda_4(t)$, given by

$$\lambda_4 = \frac{b_4(b_{11}I_1(t) + b_{22}I_2(t) + b_{33}I_3(t))}{N_h(t)}$$

and death which can occur at a rate $\mu_v = \mu_m + \mu_m 1$ where μ_m is natural death, and the death rate when in contact with ITNs given by $\mu_{m2}(\ell_1 + \ell_2 + \ell_3)$. The parameter b_4 represents the probability that a bite on human from a susceptible mosquito leads to infection of the mosquito, $b_{ii} = \frac{b_i}{\sigma_h}$. The parameter group b_{ii} also models the effect of reduced infectiousness of the middle and the high income groups. The Schematic description of the model is given in Figure 1 and the model equations are given by (2.4) and the description of the model parameters is given on Table 1.

Table 1: *Model parameters and their descriptions.*

Parameter	Description
ϵ	Average daily biting rate on man by a single mosquito Time $^{-1}$
σ_m	Probability of transmission of infection from infected humans to susceptible mosquitoes, Dimensionless
Λ_i	Recruitment rate into the i^{th} susceptible human group through birth or immigration, Time $^{-1}$
σ_h	Probability of transmission of infection from infected mosquitoes to susceptible humans, Dimensionless
μ_h	Human natural death rate, Time $^{-1}$
ρ	Number of mosquitoes per human host, Dimensionless
η_i	Efficacy of drugs in the treatment of the i^{th} infectious humans' group, Dimensionless
ν_i	Proportion of ITN usage, for the i^{th} humans' group, Dimensionless
δ_i	Disease induced death rate for the i^{th} infectious humans' group, Time $^{-1}$
z_i	ITN efficacy for the i^{th} humans' group, Time $^{-1}$
β_i	Recovery rate, for the i^{th} infections humans' group, Time $^{-1}$
γ_i	Rate at which expose human become infectious, for the i^{th} expose humans' group, Time $^{-1}$
n_s	Average number of sporozoite per mosquito, Dimensionless
α_1	Constant of proportionality, Dimensionless
α_2	Constant of proportionality, Dimensionless
θ_i	Modi cation parameter to account for reduced death rate for the i^{th} infections humans' group, Dimensionless
η_i	Measures the potency of the drug in reducing the disease, induced death rate for the i^{th} treated humans' group, Dimensionless
κ_i	Treatment rate for i^{th} infectious humans' groups, Time $^{-1}$

ζ_{max}	Maximum transmission rate, Time $^{-1}$
ϕ_i	Rate at which individuals in the i^{th} recovered humans group loses immunity, Time $^{-1}$
ζ_{min}	Minimum transmission rate, Time $^{-1}$
Λ_4	Recruitment rate into mosquito's population mosquitoes, Time $^{-1}$
μ_m	Death rate of mosquitoes Time $^{-1}$
μ_{m2}	ITNs induced death rate for mosquitoes Time $^{-1}$
u_i	Weight of the i^{th} humans' group, Dimensionless
g	Gini index, Dimensionless

$$\frac{dS_1}{dt} = \Lambda_1 - \alpha_{13}S_1S_3 + \phi_1R_1 - S_1\lambda_1 - \mu_hS_1 + \alpha_{22}S_2,$$
$$\frac{dS_2}{dt} = \Lambda_2 + \alpha_{13}S_1S_3 + R_2\phi_2 - \alpha_{22}S_2 - S_2\lambda_2 - \mu_hS_2,$$
$$\frac{dS_3}{dt} = \Lambda_3 + R_3\phi_3 - S_3\lambda_3 - \mu_hS_3,$$
$$\frac{dE_1}{dt} = S_1\lambda_1 - \gamma_1E_1 - \mu_hE_1,$$
$$\frac{dE_2}{dt} = S_2\lambda_2 - \gamma_2E_2 - \mu_hE_2,$$
$$\frac{dE_3}{dt} = S_3\lambda_3 - \gamma_3E_3 - \mu_hE_3,$$
$$\frac{dI_1}{dt} = E_1\gamma_1 + (-\beta_1 - \delta_1 - \kappa_1 - \mu_h)I_1,$$
$$\frac{dI_2}{dt} = E_2\gamma_2 + (-\beta_2 - \delta_2 - \kappa_2 - \mu_h)I_2,$$
$$\frac{dI_3}{dt} = E_3\gamma_3 + (-\beta_3 - \delta_3 - \kappa_3 - \mu_h)I_3,$$
$$\frac{dT_1}{dt} = \kappa_1I_1 - (\eta_1\beta_1 + \delta_1\theta_1 + \mu_h)T_1,$$
$$\frac{dT_2}{dt} = \kappa_2I_2 - (\eta_2\beta_2 + \delta_2\theta_2 + \mu_h)T_2,$$
$$\frac{dT_3}{dt} = \kappa_3I_3 - (\eta_3\beta_3 + \delta_3\theta_3 + \mu_h)T_3,$$
$$\frac{dR_1}{dt} = T_1\beta_1\eta_1 - R_1\mu_h - \phi_1R_1 + \beta_1I_1,$$
$$\frac{dR_2}{dt} = T_2\beta_2\eta_2 - R_2\mu_h - \phi_2R_2 + \beta_2I_2,$$
$$\frac{dR_3}{dt} = T_3\beta_3\eta_3 - R_3\mu_h - \phi_iR_3 + \beta_3I_3,$$
$$\frac{dS_m}{dt} = \Lambda_4 - S_m\lambda_4 - \mu_vS_m,$$
$$\frac{dV_m}{dt} = S_m\lambda_4 - \mu_vV_m. \qquad (2.4)$$

2.1 Basic Properties of the Model

Theorem 1 *Let the initial condition be* $S_j > 0, E_j \geq 0, I_j \geq 0, T_j \geq 0, R_j \geq 0, j = 1 \ldots 3, S_m > 0, V_m \geq 0$. *Then the solution of model (2.4) with the given initial condition will remain positive for any future time* $t > 0$.

To prove Theorem 1 we recall the second equation of model (2.4) $\frac{dS_2}{dt} = \alpha_{13}S_1S_3 + R_2\phi_2 - \alpha_{22}S_2 - \frac{S_2b_2V_m}{N_h} - \mu_hS_2 + \Lambda_2 \geq -S_2\lambda_2 - (\mu_h + \alpha_{22})S_2 + \Lambda_2$. So that

$$S_2(t) \geq \left(\int_0^t \Lambda_2 e^{\int_0^x (\lambda_2(u) + (\mu_h + \alpha_{22}))du} dx + S_2(0)\right) e^{-\int_0^t (\lambda_2(u) + (\mu_h + \alpha_{22}))du} > 0$$

Similarly, it can be shown that the other state variables $S_1 > 0, S_3 > 0, E_j \geq 0, I_j \geq 0, T_j \geq 0, R_j \geq 0, j = 1 \ldots 3, S_m > 0, V_m \geq 0$ for $t > 0$. Thus, all solutions of the model (2.4) are positive for all non-negative initial conditions, as required.

Lemma 1 *The closed set* $\Gamma = \left\{\left(S_j, E_j, I_j, T_j, R_j, S_m, V_m\right) \in \mathbb{R}_+^{17}, j = 1 \ldots 3, : N_h \leq \frac{\Lambda}{\mu_h}, N_v \leq \frac{\Lambda_4}{\mu_v}\right\}$ with $\Lambda = \Lambda_1 + \Lambda_2 + \lambda_3$, is positively-invariant and attracting for the model (2.4)

PROOF. By adding the first fifteen equations, and the remaining two equations of the model (2.4) we have

$$\frac{dN_h}{dt} = \Lambda - \mu_hN_h - \sum_{i=1}^{3}(T_i\theta_i + I_i)\delta_i,$$
$$\frac{dN_v}{dt} = \Lambda_4 - \mu_vN_v$$

respectively. The rest of the prove is similar to the proof of Lemma 1 of [30, 28]. Hence, model (2.4) is well-posed mathematically and epidemiologically in Γ and it is suffices to study the dynamics of the model in Γ, see [30, 28].

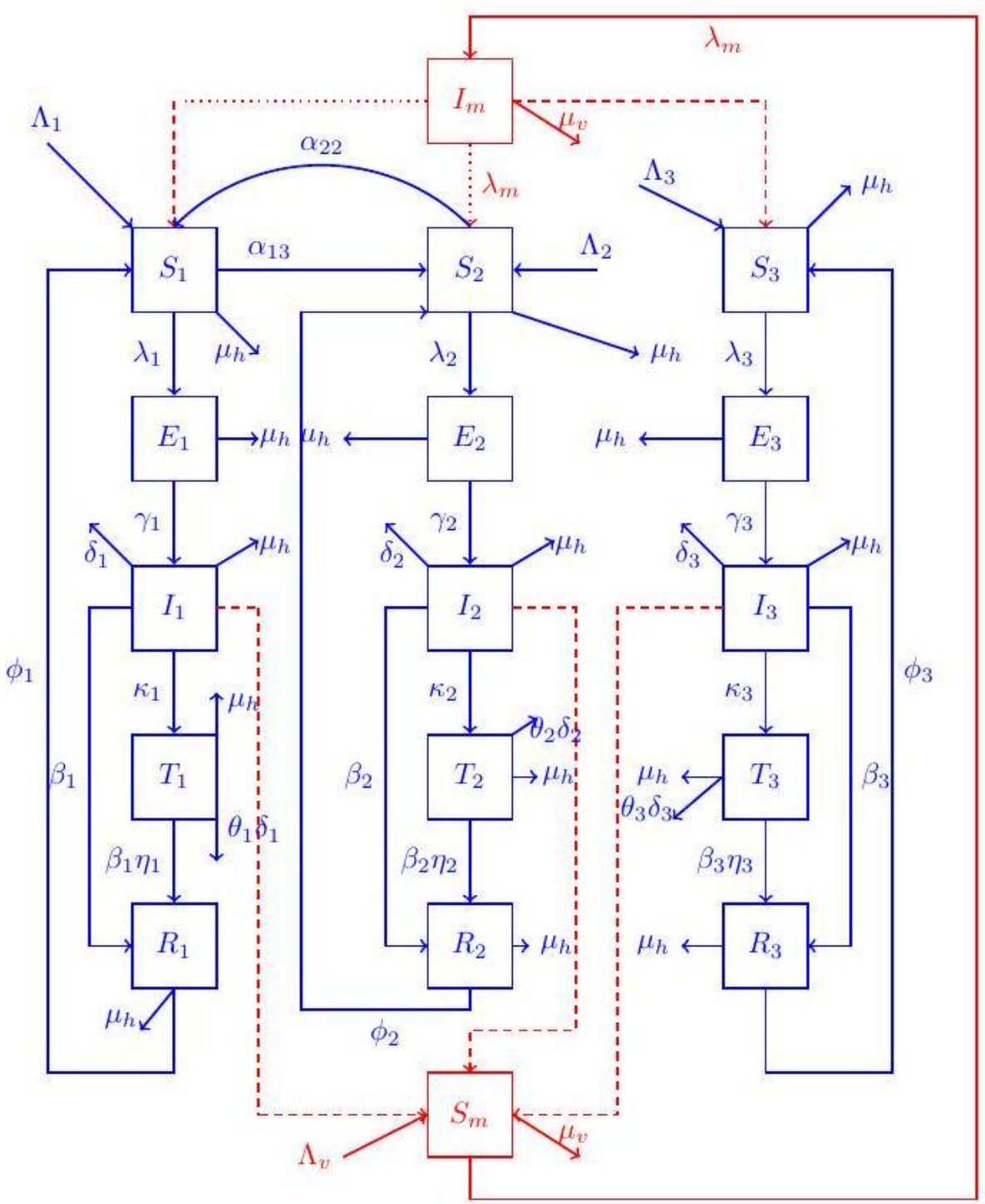

Figure 1: Schematic diagram of the model showing transfer into and out of different compartments and the corresponding transfer rate.

III. DISEASE FREE EQUILIBRIUM POINT (DFE) AND REPRODUCTION NUMBER

Model (2.4) has a DFE given by $E_{DFE} = \left(S_j^*, E_j^*, I_j^*, T_j^*, R_j^*, S_m^*, V_m^*\right) = \left(\frac{\Lambda_j}{\mu_h}, 0,0,0,0,0,0,0,0,0,0,0, \frac{\Lambda_4}{\mu_v}, 0\right), j = 1 \ldots 3$. The associated non-negative matrix F (of new infection terms) and the matrix V (of the transition terms) are given, respectively, by;

$$F=\begin{bmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{S_1^* b_1}{N_h^*} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{S_2^* b_2}{N_h^*} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{S_3^* b_3}{N_h^*} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{S_m^* b_4 b_{11}}{N_h^*} & \frac{S_m^* b_4 b_{22}}{N_h^*} & \frac{S_m^* b_4 b_{33}}{N_h^*} & 0 & 0 & 0 & 0 \end{bmatrix}$$

and,

$$V=\begin{bmatrix} J_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & J_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & J_3 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ -\gamma_1 & 0 & 0 & J_4 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -\gamma_2 & 0 & 0 & J_5 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\gamma_3 & 0 & 0 & J_6 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -\kappa_1 & 0 & 0 & J_7 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\kappa_2 & 0 & 0 & J_8 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\kappa_3 & 0 & 0 & J_9 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \mu_v \end{bmatrix}$$

respectively, where $J_k=\gamma_k+\mu_h, k=1\dots3, J_k=\beta_k+\delta_k+\kappa_k+\mu_h, k=4\dots6, J_k=\beta_k\eta_k+\ \delta_k\theta_k+\mu_h, k=7\dots9$. The dominant eigenvalue of FV^{-1} is

$$\rho_1=\frac{\sqrt{J_1J_2J_3J_4J_5J_6\mu_v S_m^* b_4\left(J_1J_2J_4J_5S_3^* b_3\gamma_3 b_{33}+J_1J_3J_4J_6S_2^* b_2\gamma_2 b_{22}+J_2J_3J_5J_6S_1^* b_1\gamma_1 b_{11}\right)}}{J_1J_2J_3J_4J_5J_6\mu_v N_h^*}$$

We define the reproduction number in relation to the DFE as

$$\Re_{eff}=\rho_1^2=\frac{S_m^* b_4\left(J_1J_2J_4J_5S_3^* b_3\gamma_3 b_{33}+J_1J_3J_4J_6S_2^* b_2\gamma_2 b_{22}+J_2J_3J_5J_6S_1^* b_1\gamma_1 b_{11}\right)}{J_1J_2J_3J_4J_5J_6\mu_v N_h^{*2}}$$

IV. BACKWARD BIFURCATION

The phenomenon of backward bifurcation, a situation where a DFE co-exists with a stable endemic equilibrium point when the reproduction is less than unity has been observed in many epidemiological models such as [30, 31]. We now investigate the condition under which bifurcation will occur in model (2.4) by choosing b_4 as the bifurcation parameter. We calculate the critical value of this parameter which makes $\Re_{eff}=1$ as

$$b_4^*=\frac{N_h^{*2}\mu_v J_1J_4J_2J_5J_3J_6}{S_4^*\left(J_1J_2J_4J_5S_3^* b_3\gamma_3 b_{33}+J_1J_3J_4J_6S_2^* b_2\gamma_2 b_{22}+J_2J_3J_5J_6S_1^* b_1 b_{11}\gamma_1\right)}. \qquad (4.1)$$

We obtained eleven eigenvalues of the Jacobian matrix of model (2.4) evaluated at the DFE and at $b_4=b_4^*$ as

$$0,-J_7,-J_8,-J_9,-J_{10},-J_{11},-J_{12},-\mu_v,-\mu_h,-\mu_h,-S_3^*\alpha_{13}-\alpha_{22}-\mu_h.$$

The remaining eigenvalues are the roots of a sextic polynomial whose coefficients are all positive and non vanishes. Thus, we have one eigenvalue with magnitude zero, and all the remaining eigenvalues have negative real parts. We let $x_i=S_i, i=1\dots3, x_i=E_i, i=4\dots6, x_i=I_i, i=\ 7\dots9, x_i=T_i, i=10\dots12, x_i=R_i, i=13\dots15, x_{16}=S_m, x_{17}=V_m$. Further more, let $\hat{f}=[f_i, i=1\dots17]$ be the left hand sides of model (2.4). The components of the right (w_i), and the left eigenvectors $(v_i), i=1\cdots17$ corresponding to the zero eigenvalue are

$$w_1=w_{16}\left(\frac{b_1x_1(\mu_h+\alpha_{22})(-\beta_1\eta_1\gamma_1\kappa_1\phi_1+J_{-}1J_4J_7J_{10}-J_7\beta_1\gamma_1\phi_1)}{J_1J_4J_7J_{10}N_h^*\mu_h(x_3\alpha_{13}+\alpha_{22}+\mu_h)}\right.$$
$$+\frac{b_2x_2\alpha_{22}(-\beta_2\eta_2\gamma_2\kappa_2\phi_2+J_2J_5J_8J_{11}-J_8\beta_2\gamma_2\phi_2)}{J_2J_5J_8J_{11}N_h^*\mu_h(x_3\alpha_{13}+\alpha_{22}+\mu_h)}$$
$$\left.-\frac{b_3x_1x_3\alpha_{13}(-\beta_3\eta_3\gamma_3\kappa_3\phi_3+J_3J_6J_9J_{12}-J_9\beta_3\gamma_3\phi_3)}{J_3J_6J_9J_{12}N_h^*\mu_h(x_3\alpha_{13}+\alpha_{22}+\mu_h)}\right),\ w_4=-\frac{x_1b_1w_{16}}{N_h^*J_1}$$
$$w_2=w_{16}\left(\frac{b_1x_1x_3\alpha_{13}(-\beta_1\eta_1\gamma_1\kappa_1\phi_1+J_1J_4J_7J_{10}-J_7\beta_1\gamma_1\phi_1)}{J_1J_4J_7J_{10}N_h^*\mu_h(x_3\alpha_{13}+\alpha_{22}+\mu_h)}\right.$$
$$+\frac{b_2x_2(x_3\alpha_{13}+\mu_h)(-\beta_2\eta_2\gamma_2\kappa_2\phi_2+J_2J_5J_8J_{11}-J_8\beta_2\gamma_2\phi_2)}{J_2J_5J_8J_{11}N_h^*\mu_h(x_3\alpha_{13}+\alpha_{22}+\mu_h)}$$

$$+\frac{b_3x_1x_3\alpha_{13}(-\beta_3\eta_3\gamma_3\kappa_3\phi_3+J_3J_6J_9J_{12}-J_9\beta_3\gamma_3\phi_3)}{J_3J_6J_9J_{12}N_h^*\mu_h(x_3\alpha_{13}+\alpha_{22}+\mu_h)}\Big), w_6=-\frac{x_3b_3w_{16}}{N_h^*J_3}$$
$$w_3=\frac{(-\beta_3\eta_3\gamma_3\kappa_3\phi_3+J_3J_6J_9J_{12}-J_9\beta_3\gamma_3\phi_3)x_3b_3w_{16}}{J_3J_6J_9J_{12}N_h^*\mu_h}, w_8=-\frac{x_2b_2\gamma_2w_{16}}{N_h^*J_2J_5},$$
$$w_{10}=-\frac{x_1b_1\gamma_1\kappa_1w_{16}}{N_hJ_1J_4J_7}, w_{11}=-\frac{x_2b_2\gamma_2\kappa_2w_{16}}{N_hJ_2J_5J_8}, w_{12}=-\frac{w_{16}x_3b_3\gamma_3\kappa_3}{J_3J_6J_9N_h}, w_5=-\frac{x_2b_2w_{16}}{N_h^*J_2},$$
$$w_{14}=-\frac{\beta_2(\eta_2\kappa_2+J_8)x_2b_2\gamma_2w_{16}}{J_2J_5J_8J_{11}N_h^*}, w_{15}=-\frac{\beta_3(\eta_3\kappa_3+J_9)x_3b_3\gamma_3w_{16}}{J_3J_6J_9J_{12}N_h^*}, w_{17}=-w_{16},$$
$$w_7=-\frac{x_1b_1\gamma_1w_{16}}{N_h^*J_1J_4}, w_9=-\frac{x_3b_3\gamma_3w_{16}}{N_h^*J_3J_6}, w_{13}=-\frac{\beta_1(\eta_1\kappa_1+J_7)x_1b_1\gamma_1w_{16}}{J_1J_4J_7J_{10}N_h^*}.$$
$$v_4=\frac{J_2J_5\gamma_1b_{11}}{J_1J_4b_{22}\gamma_2}, v_5=1, v_6=\frac{b_{33}\gamma_3J_5J_2}{J_3J_6\gamma_2b_{22}}, v_7=\frac{J_2J_5b_{11}}{J_4b_{22}\gamma_2}, v_8=\frac{J_2}{\gamma_2}, v_9=\frac{b_{33}J_5J_2}{J_6\gamma_2b_{22}},$$
$$v_{17}=\frac{J_1J_2J_4J_5x_3b_3\gamma_3b_{33}+J_1J_3J_4J_6x_2b_2\gamma_2b_{22}+J_2J_3J_5J_6x_1b_1\gamma_1b_{11}}{J_3J_6\gamma_2b_{22}J_1J_4N_h^*\mu_v}.$$

Note that $(J_1J_4J_7J_{10}-\beta_1\eta_1\gamma_1\kappa_1\phi_1-J_7\beta_1\gamma_1\phi_1)>0$, $(J_2J_5J_8J_{11}-\beta_2\eta_2\gamma_2\kappa_2\phi_2-J_8\beta_2\gamma_2\phi_2)>0, (J_3J_6J_9J_{12}-\beta_3\eta_3\gamma_3\kappa_3\phi_3-J_9\beta_3\gamma_3\phi_3>0$. The bifurcation coefficients, a and b, are given, respectively, by

$$a = \sum_{k,i,j=1}^{17} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i x_j} = \frac{2(a^+ + a^-)}{N_h^{**}},$$
$$b = \sum_{k,i=1}^{17} v_k w_i \frac{\partial^2 f_k}{\partial b_4 x_i} = \frac{v_{17}x_{16}(b_{11}w_7+b_{22}w_8+b_{33}w_9)}{N_h^{**}},$$

where

$$a^+ = s_{15}\big(v_4b_1x_1+b_2x_2+b_3v_6x_3+v_{17}b_4(b_{11}w_7+b_{22}w_8+b_{33}w_9)\big)+(x_1+x_2+x_3)v_4b_1w_1^+$$
$$a^- = (x_1+x_2+x_3)\big(-v_4b_1w_1^-+b_2w_2+b_3v_6w_3-v_{17}b_4(b_{11}w_7+b_{22}w_8+b_{33}w_9)\big)$$
$$s_{15} = \frac{ss}{J_{12}J_9J_6J_3J_{11}J_8J_5J_2J_1J_4J_7J_{10}N_h^{**}\mu_h}$$
$$ss = \gamma_1\delta_1(\kappa_1\theta_1+J_7)J_2J_3J_5J_6J_8J_9x_1b_1+\gamma_2\delta_2(\kappa_2\theta_2+J_8)b_2J_1J_3J_4J_6J_7J_9x_2$$
$$+\gamma_3\delta_3(\kappa_3\theta_3+J_9)b_3J_1J_2J_4J_5J_7J_8x_3.$$

The components of the right eigenvectors are all positive while the sign of the components of the left eigenvectors depends on the sign of w_{16} which is a free variable. If we choose $w_{16}=-1$, then $w_2<0, w_3<0$, and the remaining components of the right eigenvector are all positive except w_1 whose sign is unknown. We write $w_1=w_1^+-w_1^-$, where w_1^+, w_1^- are given by

$$w_1^+ = \frac{b_3x_1x_3\alpha_{13}(-\beta_3\eta_3\gamma_3\kappa_3\phi_3+J_3J_6J_9J_{12}-J_9\beta_3\gamma_3\phi_3)}{J_3J_6J_9J_{12}N_h^*\mu_h(x_3\alpha_{13}+\alpha_{22}+\mu_h)}$$
$$w_1^- = \frac{b_1x_1(\mu_h+\alpha_{22})(-\beta_1\eta_1\gamma_1\kappa_1\phi_1+J_1J_4J_7J_{10}-J_7\beta_1\gamma_1\phi_1)}{J_1J_4J_7J_{10}N_h^*\mu_h(x_3\alpha_{13}+\alpha_{22}+\mu_h)}$$
$$+\frac{b_2x_2\alpha_{22}(-\beta_2\eta_2\gamma_2\kappa_2\phi_2+J_2J_5J_8J_{11}-J_8\beta_2\gamma_2\phi_2)}{J_2J_5J_8J_{11}N_h^*\mu_h(x_3\alpha_{13}+\alpha_{22}+\mu_h)}$$

We simplify the bifurcation coefficient a to get $a^+>0, a^-<0$. Thus, we state the following,

Theorem 2 *Model (2.4) will under go backward bifurcation at* $\Re_{eff}=1$ *if*
$$a^+ + a^- > 0.$$

Note that the bifurcation is forward if $a^+=0$, this will happen if there no disease-induced death rate and $\alpha_{13}=0$. This means that even in the absence of a disease-induced death rate, backward bifurcation cannot be ruled out. Note that $\alpha_{13}=0$ when there is perfect income inequality $(g=1)$, hence, in the absence of disease-induced death rate and the presence of perfect income inequality, backward bifurcation will not occur in our model but can occur in the presence of imperfect income inequality $(0<g<1)$ and absence of disease-induced death rate.

V. ENDEMIC EQUILIBRIUM POINT: SPECIAL CASES

In this section we wish to find the condition under which the equilibrium point of model (2.4) exist with the disease classes non-zero. We let the endemic equilibrium point be denoted by $E_p=(S_i^{**}, E_i^{**}, I_i^{**}, T_i^{**}, R_i^{**}, S_m^{**}, V_m^{**})$, we consider two special cases;

- Model (2.4) with disease induced death rate set to zero.
- Model (2.4) with disease induced death rate set to zero and $\alpha_{13}, \alpha_{22}=0$.

5.1 Model with disease induce death rate zero

Assuming that the disease induce death rate is zero, then $N_h^{**}=\frac{\Lambda_1+\Lambda_2+\Lambda_3}{\mu_h}$..To find the equilibrium point, we set

the right hand side of model (2.4) to zero and the we expressed each equilibrium value in terms of V_m^{**} as shown in equations (5.1),(5.2) and (5.3). Finally we obtained a single equation involving only V_m^{**} given in equation (5.4).

$$\begin{aligned} E_1^{**} &= \frac{S_1^{**} b_1 V_m^{**}}{J_1 N_h^{**}}, R_1^{**} = \frac{S_1^{**} V_m^{**} b_1 \beta_1 \gamma_1 (\eta_1 \kappa_1 + J_7)}{J_1 J_4 J_7 J_{10} N_h^{**}} \\ T_1^{**} &= \frac{\kappa_1 \gamma_1 S_1^{**} b_1 V_m^{**}}{J_1 J_4 J_7 N_h^{**}}, I_1^{**} = \frac{\gamma_1 (p_9 - \Lambda_1 V_m^{**} p_4) b_1 V_m^{**} (V_m^{**} p_{13} + p_{12})}{(V_m^{**} c_3 + V_m^{**} c_2 + V_m^{**} c_1 + c_0) J_1 J_4}, \\ S_1^{**} &= \frac{N_h^{**} (-\Lambda_1 V_m^{**} p_4 + p_9)(V_m^{**} p_{13} + p_{12})}{(V_m^{**} c_3 + V_m^{**} c_2 + V_m^{**} c_1 + c_0)}. \end{aligned} \tag{5.1}$$

$$\begin{aligned} E_2^{**} &= \frac{S_2^{**} b_2 V_m^{**}}{J_2 N_h^{**}}, R_2^{**} = \frac{S_2^{**} V_m^{**} b_2 \beta_2 \gamma_2 (\eta_2 \kappa_2 + J_8)}{J_2 J_5 J_8 J_{11} N_h^{**}}, \\ T_2^{**} &= \frac{\kappa_2 \gamma_2 S_2^{**} b_2 V_m^{**}}{J_2 J_5 J_8 N_h^{**}}, I_2^{**} = \frac{(-y_2 V_m^{**2} + y_1 V_m^{**} + y_0) V_m^{**} b_2 \gamma_2}{J_5 J_2 \left(V_m^{**3} c_3 + V_m^{**2} c_2 + V_m^{**} c_1 + c_0\right)}, \\ S_2^{**} &= \frac{N_h^{**} (-\Lambda_2 V_m^{**2} p_2 p_{13} + (\Lambda_2 N_h^{**} \mu_h p_{13} - \Lambda_2 p_2 p_{12}) V_m^{**} + \Lambda_2 N_h^{**} \mu_h p_{12} + p_{11} p_{10})}{(V_m^{**} 3 + c_3 + V_m^{**} c_2 + V_m^{**} c_1 + c_0)}, \end{aligned} \tag{5.2}$$

$$\begin{aligned} E_3^{**} &= \frac{J_6 J_9 J_{12} \Lambda_3 V_m^{**} b_3}{V_m^{**} p_{13} + p_{12}}, R_3^{**} = \frac{\beta_3 (\eta_3 \kappa_3 + J_9) \Lambda_3 V_m^{**} b_3 \gamma_3}{V_m^{**} p_{13} + p_{12}}, \\ T_3^{**} &= \frac{J_{12} \Lambda_3 V_m^{**} b_3 \gamma_3 \kappa_3}{V_m^{**} p_{13} + p_{12}}, I_3^{**} = \frac{p_{14}}{V_m^{**} p_{13} + p_{12}}, \\ S_3^{**} &= \frac{p_{11}}{V_m^{**} p_{13} + p_{12}}, S_m^{**} = \frac{\Lambda_4 N_h^{**}}{I_2^{**} b_{22} b_4 + I_3^{**} b_{33} b_4 + I_1^{**} b_{11} b_4 + N_h^{**} \mu_v}. \end{aligned} \tag{5.3}$$

Here, $p_2 = \frac{b_1(\beta_1 \eta_1 \gamma_1 \kappa_1 \phi_1 - J_1 J_4 J_7 J_{10} + J_7 \beta_1 \gamma_1 \phi_1)}{J_1 J_4 J_7 J_{10}} < 0, p_4 = \frac{b_2(\beta_2 \eta_2 \gamma_2 \kappa_2 \phi_2 - J_2 J_5 J_8 J_{11} + J_8 \beta_2 \gamma_2 \phi_2)}{J_2 J_5 J_8 J_{11}} < 0$, $p_5 = N_h^{**2} \alpha_{13} \mu_h > 0, p_6 = N_h^{**} \alpha_{13} p_4 < 0, p_7 = N_h^{**} (\alpha_{22} p_2 + \mu_h p_2 + \mu_h p_4) < 0, p_8 = N_h^{**2} \mu_h (\alpha_{22} + \mu_h) > 0, p_9 = N_h^{**} (\Lambda_1 \alpha_{22} + \Lambda_1 \mu_h + \Lambda_2 \alpha_{22}) > 0, p_{10} = N_h^{**} \alpha_{13} (\Lambda_1 + \Lambda_2) > 0$, $p_{11} = J_3 J_6 N_h^{**} J_9 J_{12} \Lambda_3 > 0, p_{12} = J_3 J_6 J_9 J_{12} N_h^{**} \mu_h > 0, p_{13} = -b_3 \beta_3 \eta_3 \gamma_3 \kappa_3 \phi_3 + J_3 J_6 J_9 J_{12} b_3 - J_9 b_3 \beta_3 \gamma_3 \phi_3 > 0, p_{14} = J_9 J_{12} \Lambda_3 b_3 \gamma_3 > 0, c_0 = p_5 p_{11} + p_8 p_{12}, c_1 = p_6 p_{11} - p_7 p_{12} + p_8 p_{13} > 0$, $c_2 = p_2 p_4 p_{12} - p_7 p_{13}, c_3 = p_2 p_4 p_{13} > 0, J_{10} = \mu_h + \phi_1, J_{11} = \mu_h + \phi_2, J_{12} = \mu_h + \phi_3$, $y_0 = \Lambda_2 N_h^{**} \mu_h p_{12} + p_{11} p_{10} > 0, y_1 = \Lambda_2 N_h^{**} \mu_h p_{13} - \Lambda_2 p_2 p_{12} > 0, y_2 = \Lambda_2 p_2 p_{13} < 0.$

$$\begin{aligned} \mathrm{Y} = &(V_m^{**3} c_3 + V_m^{**2} c_2 + V_m^{**} c_1 + c_0)(\Omega_5 V_m^{**5} + \Omega_4 V_m^{**4} + \Omega_3 V_m^{**3} + \Omega_2 V_m^{**2} + \Omega_1 V_m^{**} + \Omega_0) \\ &\times (V_m^{**} p_{13} + p_{12}) \end{aligned} \tag{5.4}$$

where

$$\begin{aligned} \Omega_5 &= -\mu_v p_{13} (-J_2 J_5 \Lambda_1 b_1 b_4 b_{11} \gamma_1 p_4 p_{13} + J_1 J_2 J_4 J_5 N_h^{**} c_3 \mu_v - J_1 J_4 b_2 b_4 b_{22} \gamma_2 y_2) \\ \Omega_4 &= -J_2 J_5 b_1 b_4 b_{11} p_{13} (\Lambda_1 \Lambda_4 p_4 p_{13} - 2\Lambda_1 \mu_v p_4 p_{12} + \mu_v p_9 p_{13}) \gamma_1 \\ &\quad -J_1 J_4 b_2 b_4 b_{22} (\Lambda_4 p_{13} y_2 - \mu_v p_{12} y_2 + \mu_v p_{13} y_1) \gamma_2 \\ &\quad -J_1 J_2 J_4 J_5 \mu_v (N_h^{**} c_2 \mu_v p_{13} + N_h^{**} c_3 \mu_v p_{12} + b_4 b_{33} c_3 p_{14}) \\ \Omega_3 &= -J_2 J_5 b_1 b_4 b_{11} (2\Lambda_1 \Lambda_4 p_4 p_{12} p_{13} - \Lambda_1 \mu_v p_4 p_{12}^2 - \Lambda_4 p_9 p_{13}^2 + 2\mu_v p_9 p_{12} p_{13}) \gamma_1 \\ &\quad -J_1 J_4 b_2 b_4 b_{22} (\Lambda_4 p_{12} y_2 - \Lambda_4 p_{13} y_1 + \mu_v p_{12} y_1 + \mu_v p_{13} y_0) \gamma_2 \\ &\quad +J_1 J_2 J_4 J_5 (\Lambda_4 b_4 b_{33} c_3 p_{14} - N_h^{**} c_1 \mu_v^2 p_{13} - N_h^{**} c_2 \mu_v^2 p_{12} - b_4 b_{33} c_2 \mu_v p_{14}) \\ \Omega_2 &= -J_2 J_5 b_1 b_4 b_{11} p_{12} (\Lambda_1 \Lambda_4 p_4 p_{12} - 2\Lambda_4 p_9 p_{13} + \mu_v p_9 p_{12}) \gamma_1 \\ &\quad +J_1 J_4 b_2 b_4 b_{22} (\Lambda_4 p_{12} y_1 + \Lambda_4 p_{13} y_0 - \mu_v p_{12} y_0) \gamma_2 \\ &\quad +J_1 J_2 J_4 J_5 (\Lambda_4 b_4 b_{33} c_2 p_{14} - N_h^{**} c_0 \mu_v^2 p_{13} - N_h^{**} c_1 \mu_v^2 p_{12} - b_4 b_{33} c_1 \mu_v p_{14}) \\ &\quad = J_1 J_2 J_4 J_5 (\Lambda_4 b_4 b_{33} c_2 p_{14} - N_h^{**} c_0 \mu_v^2 p_{13} - N_h^{**} c_1 \mu_v^2 p_{12} - b_4 b_{33} c_1 \mu_v p_{14}) \\ &\quad +J_1 J_2 J_4 J_5 (\Lambda_4 b_4 b_{33} c_1 p_{14} - N_h^{**} c_0 \mu_v^2 p_{12} - b_4 b_{33} c_0 \mu_v p_{14}) \\ \Omega_0 &= \Lambda_4 b_4 J_1 J_2 J_4 J_5 b_{33} c_0 p_{14}. \end{aligned}$$

The factors $(V_m^{**3} c_3 + V_m^{**2} c_2 + V_m^{**} c_1 + c_0)$ and $(V_m^{**} p_{13} + p_{12})$ have all positive coefficients, thus by Descartes rule of sign, non of them have any positive root. Hence, the positive roots of equation (5.4) are the positive roots of the quintic

$$\Omega_5 V_m^{**5} + \Omega_4 V_m^{**4} + \Omega_3 V_m^{**3} + \Omega_2 V_m^{**2} + \Omega_1 V_m^{**} + \Omega_0 = 0. \tag{5.5}$$

We are unable to determine the signs of $\Omega_i, i = 1 \ldots 4$ but we find that $\Omega_5 < 0, \Omega_0 > 0$. Therefore, we have at least one sign change in the coefficients of the quintic given in equation 5.5. Hence, by Descartes' rule of sign, equation (5.5) has at least one positive root. Note that each of the terms in systems (5.1) to (5.3) is positive. So that

for each positive root of equation (5.5) the existence of positive equilibrium values of other state variables is guaranteed. Thus, we state the following;

Theorem 3 *Model (2.4) with disease induced death rate set to zero has at least one endemic equilibrium point whose existence does not necessarily depend on the reproduction number* $\Re_{eff}$*, moreover, the number of endemic equilibrium points is same as the number of positive roots of the quintic given in equation (5.5).*

The implication of Theorem 3 is that, the classification of humans into various epidemiological groups considered in this report may lead to multiple steady states that are not typical of many malaria models. This is because backward bifurcation can still occur even when the disease induced-death rate is set to zero.

5.2 Endemic Equilibrium point: Special Case 2

In this subsection, we wish to find the endemic equilibrium point in a situation where the disease-induced death rate is zero and that susceptible individuals with low income do not join the susceptible individuals with middle income and vice-versa $(\alpha_{13} = 0, \alpha_{22} = 0)$. By following a procedure similar that in subsection ??, we find that the equilibrium point of model (2.4) with $\delta_i = 0, \alpha_{13} = 0, \alpha_{22} = 0$ depends on the positive roots of the cubic

$$\Phi = \omega_3 V_m^{**3} + \omega_2 V_m^{**2} + \omega_1 V_m^{**} + \omega_0 \tag{5.6}$$

where,

$$\begin{aligned}\omega_3 \quad &= J_1 J_3 J_4 J_6 N_h^{**} \mu_v p_2 p_{13} (J_2 J_5 N_h^{**} \mu_v p_4 - \Lambda_2 b_2 b_4 b_{22} \gamma_2) \\ &+ J_2 J_5 b_4 \mu_v p_4 (-J_3 J_6 \Lambda_1 N_h^{**} b_1 b_{11} \gamma_1 p_{13} + J_1 J_4 b_3 b_{33} \gamma_3 p_2 p_{11})\end{aligned}$$

$\omega_0 = (1 - \Re_{eff}) J_1 J_2 J_3 J_4 J_5 J_6 N_h^4 \mu_h^2 \mu_v^2 p_{12}$. Note that $\omega_3 > 0$, and $\omega_0 > 0$ if $\Re_{eff} < 1$. We state the following results

Theorem 4 *Model (2.4) with disease induced death rate set to zero and* $\alpha_{13} = 0, \alpha_{22} = 0$*, has*

1. At least one and at most three endemic equilibrium points if $\Re_{\text{eff}} > 1$,
2. Zero or two endemic equilibrium points if $\Re_{eff} < 1$.

To prove the first part of Theorem 4, we note that $\Phi(0) < 0$, if $\Re_{eff} > 1$ and $\lim_{V_m \to \infty} \Phi(V_m) = \infty$ since the leading term is positive. Thus, by intermediate value theorem, a positive root of $\Phi(V_m)$ exist. By considering the signs pattern of the coefficients of $\Phi(V_m)$, we find that there is at least one sign change and at most three sign changes. The results follows from Descartes' rule of sign. To prove the second part, we find that there are only two possibilities for the signs pattern of the coefficients of $\Phi(V_m)$ for $\Re_{eff} < 1$. These are two sign changes and one sign preservation or three signs preservations and no sign change. Hence, from Descartes' rule of sign the number of positive roots are either 0 or two.

VI. NUMERICAL SIMULATION

We conducted numerical simulation of model (2.4) by first assigning weights to each income group as $u_1 = 0.4, u_2 = 0.75, u_3 = 0.97$, for low, middle and high income groups respectively. The other parameter values as given on Table 2. The results of the simulations are presented on Figures 2, 3 and 4 using the initial conditions indicated. The results in Figure 2 are for the three different groups of susceptibles using different values of the Gini index. It can be seen that over a small period of time, smaller income inequality (smaller values of g) will result in higher growth of the susceptible with low income. The negative impacts of inequality manifest themselves in the long run where we notice a high growth rate for susceptible individuals with low income. Since this group contains the lowest income earners, this implies that high income inequality will lead to an increase in the number of people that finds it difficult to afford personal protection against malaria and its treatment.

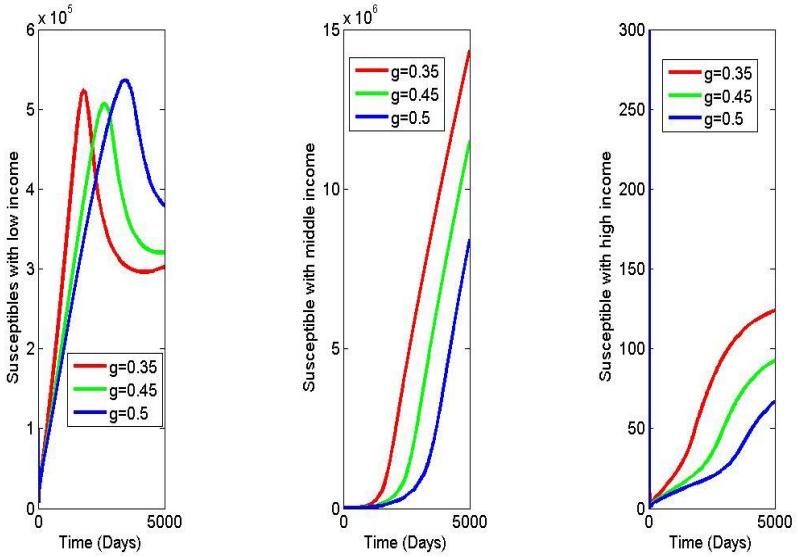

Figure 2: Numerical simulation of model three (2.4) for different susceptible humans using the initial condition $(S_1, S_2, S_3, E_1, E_2, E_3, I_1, I_2, I_3, T_1, T_2, T_3, R_1, R_2, R_3, S_m, V_m) = (10^5, 5 \times 10^3, 3 \times 10^2, 500, 30, 20, 103, 5 \times 102, 50, 70, 40, 32, 600, 10, 10, 2 \times 105, 12 \times 10^4)$ *and the parameter values given on Table with the various values of Gini index as shown.*

As for susceptible with middle and high incomes, it can be seen from the middle and the third panels of Figure 2 that the lower the inequality, the higher the growth of the population being simulated. The dynamics in the middle panel of Figure 2 might be explained by the fact that when the Gini index is high, more individuals from susceptible group 2 will move to susceptible group 1.

Table 2: *Parameter values used in the simulation and their sources.*

Parameter	**Value**	Source
ϵ	0.1991	[60]
σ_m	0.83	[12]
Λ_i	$\Lambda_1 = 4000, \Lambda_2 = 100, \Lambda_3 = 0.001,$	Assumed
σ_h	0.5	[12]
μ_h	3.91×10^{-5}	[12]
ρ	8	[31]
η_i	Variable	Assumed
ν_i	Variable	Assumed
δ_i	$\delta_1 = 0.0083, \delta_2 = 0.8\delta_1, \delta_3 = 0.4\delta_1$	Assumed
z_i	$z_1 = 0.3, z_2 = 0.65, z_3 = 0.97$	Assumed
β_i	$\beta_1 = 1/285, \beta_2 = \beta_1, \beta_3 = \beta_1$	[12]
γ_i	$\gamma_1 = 1/14, \gamma_2 = \gamma_1, \gamma_3 = \gamma_1$	[12]
n_s	3	Assumed
α_1	0.0005	Assumed
α_2	0.0005	Assumed
θ_i	Variables	Assumed
η_i	Variables	Assumed
k_i	Variable	Assumed
ζ_{max}	0.9	[46]
ϕ_i	$\phi_1 = 5.4795 \times 10^{-4}, \phi_2 = \phi_1, \phi_3 = \phi_1$	[46]
ζ_{min}	0.0696	[46]
Λ_4	6000	Assumed
μ_m	0.04	[12]
μ_{m2}	0.0995	[31]

u_i	$u_1 = 0.4, u_2 = 0.75, u_3 = 0.97$	Assumed
g	Variable	Assumed

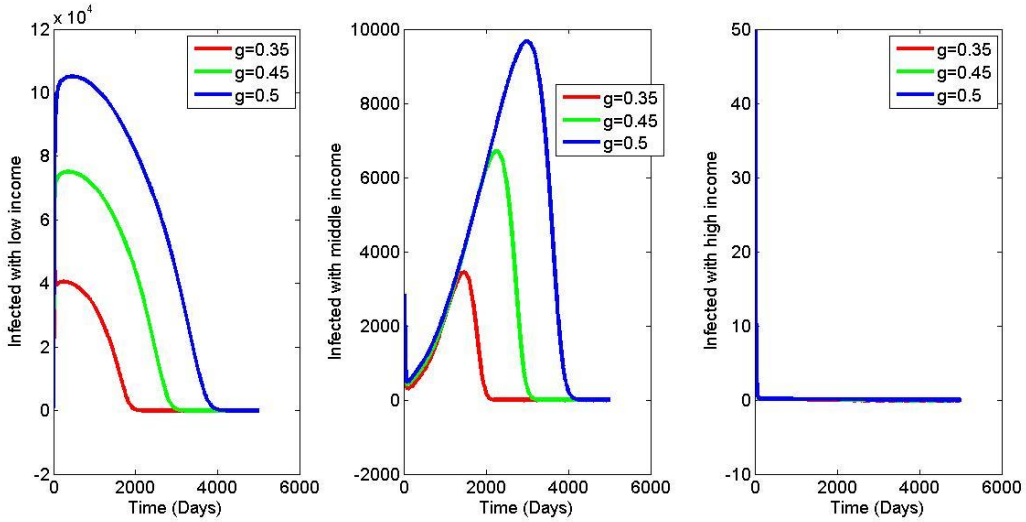

Figure 3: Numerical simulation of model three (2.4) for different susceptible humans using the initial condition $(S_1, S_2, S_3, E_1, E_2, E_3, I_1, I_2, I_3, T_1, T_2, T_3, R_1, R_2, R_3, S_m, V_m) = (10^5, 5 \times 10^3, 3 \times 10^2, 500, 30, 20, 103, 5 \times 102, 50, 70, 40, 32, 600, 10, 10, 2 \times 105, 12 \times 10^4)$ *and the parameter values given on Table with the various values of Gini index as shown.*

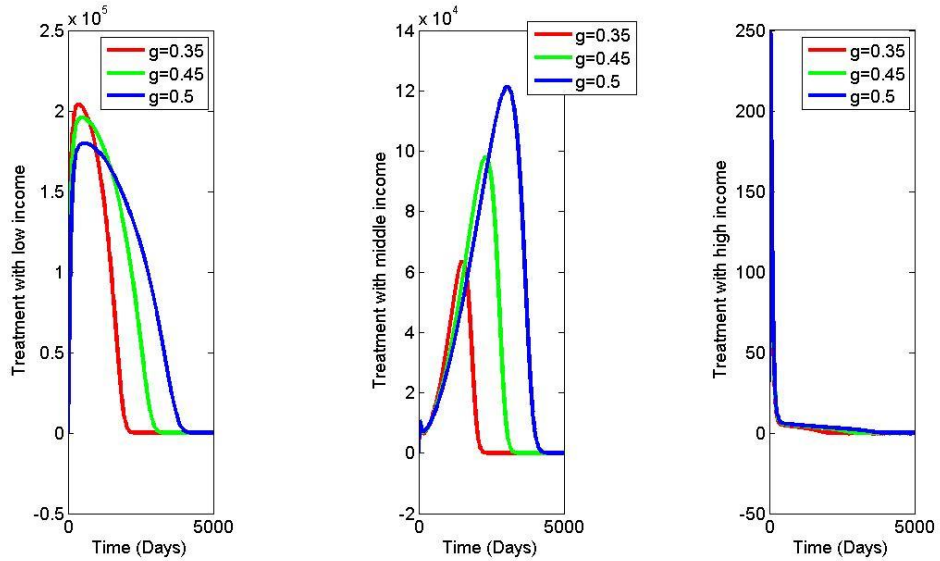

Figure 4: Numerical simulation of model three (2.4) for different susceptible humans using the initial condition $(S_1, S_2, S_3, E_1, E_2, E_3, I_1, I_2, I_3, T_1, T_2, T_3, R_1, R_2, R_3, S_m, V_m) = (10^5, 5 \times 10^3, 3 \times 10^2, 500, 30, 20, 103, 5 \times 102, 50, 70, 40, 32, 600, 10, 10, 2 \times 105, 12 \times 10^4)$ *and the parameter values given on Table with the various values of Gini index as shown.*

From the first and the second panels of Figure 3 it can be seen that the higher the number of people who become infected, the longer it takes for the disease to be eradicated. From the third panels of Figures 3 and 4 one can say that the values of the Gini indexes used do not affect the number of infections or treated individuals with high income. From the first and second panels of Figure 4 one can see that the number of individuals in the treatment compartments does not vary much for the different Gini indexes over a small period of time. As time progresses, the results become similar to those shown in Figure 3.

The results indicate that the policy of countering growing income inequality will be beneficial to the population in line with the work in [43]. Reducing income-inequality is not only beneficial to population growth but helps in the number of malaria cases and hence, the number of people being hospitalized. In addition to the disease-induced death rate, backward bifurcation can also appear as a result of the movement of susceptible individuals with low income, to susceptible individuals with middle income.

VII. CONCLUSION

In this report, we formulated a deterministic mathematical model of malaria transmission by dividing the total human population broadly into three based on income level. We calculated the reproduction number and found the condition under which backward bifurcation may occur in the model. Our analysis indicates that even without a disease induce-death rate, backward bifurcation may occur in the model. The numerical simulation conducted shows that widening income inequality will increase malaria cases and the number of people being hospitalized.

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