

Theoretical Study of a Model for Dengue and its Co- Endemicity with Chikungunya Virus

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Article Info Abstract

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A deterministic nonlinear mathematical model describing the population dynamics for Dengue and Chikungunya virus taken into consideration the effect of misdiagnosis due to the co-endemicity of the two viruses in the human population. It is necessary to understand the most important parameters involved in their dynamics that may help in the development strategies for prevention, control, and joint treatments. The model is rigorously analyzed qualitatively and thresholds for eradication are established.

1. Introduction

The Chikungunya (CHIKV) is known to be a genus of single-stranded virus that infect humans, which cause acute febrile polyarthralgia and arthirtis [1]. The manifestation of Chikungunya illness are fever and joint pain, other symptoms may include headache, muscle pain, joint swelling and skin rash [2]. Chikungunya virus can be found in the blood of an infected human, passed from an infected person to a mosquito through the bites of an Aedes Spp. mosquitoes [2]. Chikungunya virus (CHIKV) has an intrinsic incubation period in a human host of about four days after an infected mosquito bite, and the illness lasts a period of seven days approximately. It is during this period that mosquitoes can be infected with CHIKV [3]. CHIKV outbreaks in Africa and Asia have been reported since the 1960s, the major activity which took place from 1960s to 1980s lead to the spread of the virus infection to other regions [4]. CHIKV has spread quickly to several islands in Indian Ocean, to Indian and to Southeast Asia [5]. The epizootic life cycle of CHIKV in Africa is poorly understood

and that of Nigeria is under reported. Furthermore, a few segregation of CHIKV have been documented concerning other mammalian species (example, bat and squirrels) [6].

Dengue virus is an eruptive febrile illness transmitted by arthropods called the Aedes aegypti, which causes Dengue fever [7]. The symptoms of Dengue disease are persistent vomiting, severe abdominal ache, joint pain, and headache and skin rash [8]. Those who become infected with the virus a second time develop severe disease [9]. The severe stage of Dengue is respiratory insufficiency, severe bleeding, altered conscious, heart liver and dengue shock syndrome (DSS) which can lead to death [10, 11]. The symptoms of Chikungunya are similar to those of Dengue disease because they are spread by the same Aedes mosquitoes [2]. Aedes aegypti is one of the main vector that has been recognize to be the cause of the virus, and the male A. aegypti mosquitoes was tested to be positive for the transmission of Chikungunya [12]. Dengue and Chikungunya virus co-circulating have been announced to exist in 98 countries to which the viruses are widely distributed and are presumed to take place in regions with high transmission force [13]. The countries that currently experienced unusual number of Dengue infection that coincide with that of Chikungunya infection is the Americas and a total of 2.3million DENV infections and 635,000 CHIKV infections were reported in this region in 2015 [14]. Several models have been made to study the transmission dynamics of Dengue and Chikungunya virus, our study is focus on the misdiagnosis of the disease due to the co-endemicity of the two viruses in the human population.

2.0 Model Formulation

The model, to be designed, assumes homogeneous mixing of the human and vector (mosquito) population, so that each mosquito bite has equal chance of transmitting the virus to a susceptible human in the population because only an aegypti has been reported to transmit both viruses. It is imperative that we state at this juncture that the model we are formulating here is not a co-infection model but a co-circulating model that takes account of misdiagnosis when treatment is carried out in a patient. Hence, the total human population at any time tis denoted by $N_H(t)$, is partitioned into eleven mutually-disjoint compartments of susceptible humans $(S_H(t))$, population of susceptible Dengue vectors $(S_{MD}(t))$ and population of susceptible Chikungunya vectors $(S_{MC}(t))$, population of exposed humans and vectors (Dengue and Chikungunya) $(E_D(t))$, $(E_{MD}(t))$ and $(E_{MC}(t))$, infectious humans with Dengue $(I_{D_1}(t))$, infectious humans with Chikungunya $(I_{C_1}(t))$, population of human with Dengue and Chikungunya diagnosed $(I_{D2}(t))$ and $(I_{C2}(t))$ population of human wrongly diagnosed with Dengue and Chikungunya $(I_{DW}(t))$ and Population of recovered humans from Dengue and Chikungunya^{$(R_D(t))$}, population of infectious vectors with Dengue $(I_{MD}(t))$ and population of infectious vectors with Chikungunya $(I_{MC}(t))$, so that

$$
N_H(t) = S_H(t) + E_D(t) + I_{D1}(t) + I_{D2}(t) + I_{WD}(t) + R_D + I_{C1}(t) + I_{C2}(t) + I_{WC}(t) + R_C(t)_h
$$
 (1)

Similarly, the vector populace at time *t*, is fragmented into susceptible Dengue vectors $(S_{MD}(t))$, exposed vectors with Dengue $(E_{MD}(t))$, infectious vectors with Dengue $(I_{MD}(t))$, susceptible Chikungunya vectors $(S_{MC}(t))$, exposed vectors with Chikungunya $(F_{MC}(t))$, infectious vectors with Chikungunya $(I_{MC}(t))$.

The susceptible human populace is assumed to be engendered via enrollment at the frequency A_H . This populace diminished as susceptible individuals make contact with infected vectors, the force of infection with Dengue is at the frequency λ_{DV} and the force of infection with Chikungunya is at the frequency λ_{CV} , where:

$$
\lambda_{CV} = \frac{\beta_{CV}b_{CV}(\eta_{C1}E_C + I_{C1} + \eta_{C2}I_{C2} + I\eta_{C3}I_{WC})}{N_H}
$$
\n
$$
\lambda_{DV} = \frac{\beta_{DV}b_{DV}(\eta_{D1}E_C + I_{D1} + \eta_{D2}I_{D2} + I\eta_{D3}I_{WD})}{N_H}
$$
\n(2)

Here, β_{DV} is the frequency of Dengue transmission from vectors to humans and β_{CV} is the rate of Chikungunya transmission from vectors to humans The modification parameter η_{D1} is the parameter for reduced infectiousness of humans exposed to dengue, η_{D2} is the modification parameter that account for less infectiousness of humans rightly diagnosed for dengue and η_{D3} is the modification parameter for increased infectiousness of humans wrongly diagnosed for dengue. The modification parameter η_{C1} is the parameter for reduced infectiousness of humans exposed to Chikungunya, η_{c2} is the modification parameter for reduced infectiousness of humans rightly diagnosed for Chikungunya and η_{C3} is the modification parameter for increased infectiousness of humans wrongly diagnosed for Chikungunya, hence the assumption $0 < \eta_{D1} < \eta_{D2} < \eta_{C1} < \eta_{C2} < 1$ for reduced infectiousness and $0 > \eta D3 > \eta C3 > 1$ $0 > \eta_{D3} > \eta_{C3} > 1$ for increased infectiousness of humans wrongly diagnosed for either of the diseases [15]. Similarly, it can be revealed that the frequency at which vectors becomes infected is given by λ_{DH} (the force of infection for vectors with Dengue) and λ_{CH} (the force of infection for vectors with Chikungunya) where

$$
\lambda_{DH} = \frac{\beta_{DH}b_{DV}I_{MD}}{N_H}, \quad and \quad \lambda_{CH} = \frac{\beta_{CH}b_{CV}I_{MC}}{N_H}
$$
(3)

 β_{DH} is the Probability transmission of Dengue from vectors to humans and b_{DV} is the biting frequency of vectors that transmit Dengue, also β_{CH} is the probability transmission of Chikungunya from vectors to humans and b_{CV} is the biting frequency of vectors that transmit Chikungunya. Individuals in each compartment suffer normal mortality frequency $^{\mu_H}$. The frequency of change of the susceptible human populace is generated by recruitment at the frequency Λ ^{$_H$}. This populace decreases as they come in contact with vectors infectious with Dengue at the frequency λ_{DH} , with vectors infectious with Chikungunya at the

frequency
$$
\lambda_{CH}
$$
, and finally by usual mortality at the frequency μh . Thus
\n
$$
\frac{dS_H}{dt} = \Lambda_H - \lambda_{DH} S_H - \lambda_{CH} S_H - \mu_H S_H,
$$
\n(4)
\nThe population of infected individual with Dengue is generated by new infecti

The population of infected individual with Dengue is generated by new infection at the frequency of λ_{DH} , and diminish by progression of individuals (at the frequency λ_{DH}) and (at the frequency μ ^{*H*} μ </sup>). Thus, equ
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$$
\frac{dE_D}{dt} = \lambda_{DH} S_H - (\gamma_D + \mu_H) E_D,\tag{5}
$$

The populace of infectious humans with Dengue is produced by the progression of infected humans with Dengue (at the frequency $\binom{\gamma_D}{D}$). This populace diminishes due to diagnoses (at the frequency θ_p), normal mortality (at the frequency μ ^H) and dengue prompted mortality (at the frequency δ_p). So that; eq
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$$
\frac{dI_{D1}}{dt} = \gamma_D E_D - (\delta_D + \mu_H + \theta_D) I_{D1},
$$
\n(6)

The populace of infectious humans with Dengue that are correctly diagnosed is generated by inflow of individual from I_{D1} class (at the frequency $\theta_D(1-P_D)$) while $(1-P_D)$ represent the remaining fraction of humans wrongly diagnosed for dengue. This populace decreases due to recovery from Dengue (at the frequency $^{\tau_p}$), normal mortality (at the frequency $^{\mu_{H}}$) and Dengue-prompted mortality (at the frequency $\alpha_D \delta_D$) where α_D is the parameter that account for reduced mortality of humans rightly diagnosed for dengue; so that ue
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$$
\frac{dI_{D2}}{dt} = \theta_D (1 - P_D) I_{D1} - (\alpha_D \delta_D + \tau_D + \mu_H) I_{D2} - \phi_D I_{WD},\tag{7}
$$

The population of infectious humans that are wrongly diagnosed of Dengue cases is generated by inflow of individual from I_{D1} class at the frequency $\phi_D P_D$) while P_D represent the fraction of humans wrongly diagnosed for dengue. This population decreases due to rediagnosed for Dengue (at the frequency $\stackrel{\phi_D}{\sim}$), natural death (at the frequency $\stackrel{\mu_H}{\sim}$) and dengueinduced death (at the frequency δ_p); Thus, ag
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$$
\frac{dI_{WD}}{dt} = \phi_D P_D I_{D1} - (\mu_H + \delta_D + \phi_D) I_{WD},
$$
\n(8)

The recovered populace from Dengue is generated by humans from infectious class (at the frequency $^{\tau_{D}}$). This population is reduced by natural death (at the frequency $^{\mu_{H}}$). Thus, re

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$$
\frac{dR_D}{dt} = \tau_D I_{D2} - \mu_H R_D,\tag{9}
$$

The population of infected individual with Chikungunya is generated by new infection at the frequency of λ_{CH} , and decreases by progression of individuals (at the frequency γ_{H}) and natural death (at the frequency $^{\mu_H}$). Thus,

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$$
\n
$$
\frac{dE_C}{dt} = \lambda_{CH} S_H - (\gamma_C + \mu_H) E_C,
$$
\n(10)
\nThe populace of infectious humans with Chikungunya is generated by the progression

The populace of infectious humans with Chikungunya is generated by the progression of expose humans with Chikungunya (at the frequency ℓ^2). This populace decreases due to diagnoses (at the frequency θ_c), natural death (at the frequency μ ^H) and Chikungunya induced death (at the frequency δ_c). So that iag
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$$
\frac{dI_{C1}}{dt} = \gamma_C E_C - (\delta_C + \mu_H + \theta_C)I_{C1},
$$
\n(11)

The populace of infectious humans with Chikungunya that are correctly diagnosed is generated by inflow of individual from I_{c1} class (at the frequency $\theta_c(1-P_c)$) while $(1-P_c)$ represent the remaining fraction of humans wrongly diagnosed for Chikungunya. This population decreases due to recovery from Chikungunya (at the frequency ^{τ_c}), natural death (at the frequency $^{\mu_{H}}$) and Chikungunya-induced death (at the frequency $^{\alpha_{C}\delta_{C}}$) where $^{\alpha_{C}}$ is the parameter that account for reduced mortality of humans rightly diagnosed for Chikungunya; so that at
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the parameter that account for reduced mortality of humans rightly diagnosed for
Chikungunya; so that

$$
\frac{dI_{C2}}{dt} = \theta_C (1 - P_C) I_{C1} - (\alpha_C \delta_C + \tau_C + \mu_H) I_{C2} - \phi_C I_{WC},
$$
(12)

The population of infectious humans that are wrongly diagnosed of Chikungunya cases is generated by inflow of individual from I_{c1} class at the frequency $\theta_c P_c$) while P_c represent the fraction of humans wrongly diagnosed for Chikungunya. This population decreases due to re-diagnosed for Chikungunya (at the frequency ϕ_c), natural death (at the frequency μ ^H) and Chikungunya-induced death (at the frequency δ_c); Thus, re-c
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Chikungunya-induced death (at the frequency
$$
\delta_c
$$
); Thus,
\n
$$
\frac{dR_c}{dt} = \tau_c I_{c2} - \mu_H R_c,
$$
\n(13)

Populace of susceptible mosquitoes are increase by birth (at the frequency Λ_{ν}) and decrease by infection, with infected humans with Chikungunya (at the frequency λ_{DV}) and natural death (at the frequency μ ^v). Thus, f i
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$$
\frac{dS_{MD}}{dt} = \Lambda_{VD} - \lambda_{VD} S_{MD} - \mu_V S_{MD},\tag{14}
$$

The infected vector population with Dengue is generated via the infection of susceptible vector with Dengue (at the frequency Λ_{ν}), the populace is diminished by natural death (at the frequency $^{\mu_V}$) and progression of vectors from exposed to infectious (at the frequency $^{\lambda_{MD}}$). This gives:

$$
\frac{dE_{MD}}{dt} = \lambda_{VD} S_{MD} - (\gamma_{MD} + \mu_V) E_{MD},\tag{15}
$$

The infectious vector population with Dengue virus is generated via the progression of vectors from E_{MC} class (at the frequency γ_{MD}) and it is reduced by natural death (at the frequency μ_V). This gives: ect
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$$
\frac{dI_{MD}}{dt} = \gamma_{MD} E_{MD} - \mu_V I_{MD},\tag{16}
$$

Susceptible mosquitoes Populace with Chikungunya virus are increase by birth (at the frequency $\Lambda_{\nu c}$) and decrease by infection with infected humans with Chikungunya (at the frequency λ_{ν}) and natural death (at the frequency μ_{ν}). Thus,

$$
\frac{dS_{MC}}{dt} = \Lambda_{VC} - \lambda_{VC} S_{MC} - \mu_V S_{MC},\tag{17}
$$

The infected vector population with Chikungunya is generated via the infection of susceptible vector with Chikungunya (at the frequency λ ^{χ}), the populace is diminished by natural death (at the frequency μ ^V $_{V}$) and progression of vectors (at the frequency γ _{MC}). This gives: sc
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$$
\frac{dE_{MC}}{dt} = \lambda_{VC} S_{MC} - (\gamma_{MC} + \mu_V) E_{MC}
$$
\n(18)

The infectious vector population with Chikungunya is generated via the progression of vectors from exposed class (at the frequency γ_{MC}) and it is reduced by natural death (at the frequency μ ^v). This gives: *dl*
dl

frequency
$$
\mu_V
$$
). This gives:
\n
$$
\frac{dI_{MC}}{dt} = \gamma_{MC} E_{MC} - \mu_V I_{MC}.
$$
\n(19)
\nHence, the Dengue Chikungunya transmission model comprises of the following system of

Hence, the Dengue Chikungunya transmission model comprises of the following system of 17 nonlinear ordinary differential equations. The schematic is show in Figure 1.

Figure 1: Schematics representation for the basic Dengue Chikungunya model

$$
\frac{dS_H}{dt} = \Lambda_H - (\lambda_{DH} + \lambda_{CH} + \mu_H)S_H,
$$
\n
$$
\frac{dE_D}{dt} = \lambda_{DH}S_H - (\gamma_D + \mu_H)E_D,
$$
\n
$$
\frac{dI_{D1}}{dt} = \gamma_D E_D - (\delta_D + \mu_H + \theta_D)I_{D1},
$$
\n
$$
\frac{dI_{D2}}{dt} = \theta_D (1 - P_D)I_{D1} - (\alpha_D \delta_D + \tau_D + \mu_H)I_{D2} - \phi_D I_{WD},
$$
\n
$$
\frac{dI_{WD}}{dt} = \phi_D P_D I_{D1} - (\mu_H + \delta_D + \phi_D)I_{WD},
$$
\n
$$
\frac{dR_D}{dt} = \tau_D I_{D2} - \mu_H R_D,
$$
\n
$$
\frac{dE_C}{dt} = \lambda_{CH}S_H - (\gamma_C + \mu_H)E_C,
$$
\n
$$
\frac{dI_{C1}}{dt} = \gamma_C E_C - (\delta_C + \mu_H + \theta_C)I_{C1},
$$
\n
$$
\frac{dI_{C2}}{dt} = \theta_C (1 - P_C)I_{C1} - (\alpha_C \delta_C + \tau_C + \mu_H)I_{C2} - \phi_C I_{WC},
$$
\n
$$
\frac{dI_{WC}}{dt} = \phi_C P_C I_{C1} - (\mu_H + \delta_C + \phi_C)I_{WC},
$$
\n
$$
\frac{dS_{MD}}{dt} = \tau_C I_{C2} - \mu_H R_C,
$$
\n
$$
\frac{dS_{MD}}{dt} = \lambda_{VD} - \lambda_{VD} S_{MD} - \mu_V S_{MD},
$$
\n
$$
\frac{dI_{MD}}{dt} = \gamma_{MD} S_{MD} - (\gamma_{MD} + \mu_V)E_{MD},
$$
\n
$$
\frac{dI_{MC}}{dt} = \lambda_{VC} - \lambda_{NC} S_{MC} - \mu_V S_{MC},
$$
\n
$$
\frac{dI_{MC}}{dt} = \lambda_{VC} S_{MC} - (\gamma_{MC} + \mu_V)E_{MC},
$$
\n(20)

Table 2: Description of parameters of model (20)

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3.0 Mathematical Analysis

For the impact of misdiagnosis of Dengue for Chikungunya and vice versa on the dynamics of both diseases in the population with minimal co-infection to be epidemi- ologically and biologically meaningful, it is important to prove that all trajectories with positive initial data remain positive for all time and the feasible region will also remain positively-invariant for all time.

Theorem 1 Let the initial figures for the dengue and Chikungunya model (20) be given as $S_H(0) > 0$, $E_D(0) > 0$, $I_{D1}(0) > 0$, $I_{D2}(0) > 0$, $I_{WD}(0) > 0$, $R_D(0) > 0$, $E_C(0) > 0$, $I_{C1}(0) > 0$, $I_{C2}(0) > 0$, $I_{WC}(0) > 0$, $R_C(0) > 0$, $S_{MD}(0) > 0$, $E_{MD}(0) > 0$, $I_{MD}(0) > 0$, $S_{MC}(0) > 0$, and $I_{MC}(0) > 0$ _.

$$
(s_H(t), E_D(t), I_{D1}(t), I_{D2}(t), I_{DW}(t), R_D(t), S_H(t), E_C(t), I_{C1}(t),
$$

Then the trajectories $I_{C2}(t), I_{WC}(t), R_C(t), S_{MD}(t), E_{MD}(t), I_{MD}(t), S_{MC}(t), E_{MC}(t), I_{MC}(t))$

of the model with positive initial conditions, will remain positive for all time *t >* 0.

of the model with positive initial conditions, will remain positive for all time
$$
t > 0
$$
.
\n**Proof:** Following [16] and [17], the model (20) can be written in the form\n
$$
\frac{dY}{dt} = P(Y)Y + G
$$
\n(21)

$$
Y = (S_H(t), E_D(t), I_{D1}(t), I_{D2}(t), I_{WD}(t), R_D(t), E_C(t), I_{C1}(t), I_{C2}(t),
$$

$$
I_{WC}(t), R_C(t), S_{MD}(t), E_{MD}(t), I_{MD}(t), S_{MC}(t), E_{MC}(t), I_{MC}(t))
$$

where

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(0, 0) , *^T G* = *^H*

and $P(Y)$, is a 17 x 17 matrix given as

$$
f(Y) = (f_{1(17x10)} - f_{2(17x7)})
$$
(22)
\n
$$
p_1 = \frac{(\beta_{DH}b_{DV}Y_{14} + \beta_{CH}b_{CV}Y_{17})}{N_H}, \qquad p_2 = \frac{(\beta_{DV}b_{DV}(\eta_{D1}Y_2 + Y_3 + \eta_{D2}Y_4 + Y_5))}{N_H}
$$
(22)
\nWhere
\n
$$
p_3 = \frac{(\beta_{CV}b_{CV}(\eta_{C1}Y_7 + Y_8 + \eta_{C2}Y_9 + \eta_{C3}Y_{10})}{N_H}, \qquad g_1 = \gamma_D + \mu_H, g_2 = \delta_D + \mu_H + \theta_D, g_3 = \alpha_D\delta_D + \tau_D + \mu_H,
$$
\n
$$
g_2 = \delta_D + \phi_C + \mu_U, g_4 = \delta_C + \mu_U + \theta_C, g_5 = \alpha_C\delta_C + \tau_C + \mu_U, g_6 = \delta_C + \phi_C + \mu_U
$$

$$
g_4 = \delta_D + \phi_C + \mu_H, \quad g_5 = \gamma_{MC} + \mu_H, g_6 = \delta_C + \mu_H + \theta_C, g_7 = \alpha_C \delta_C + \tau_C + \mu_H, g_8 = \delta_C + \phi_C + \mu_H, g_9 = \gamma_{MD} + \mu_V, g_{10} = \gamma_{MC} + \mu_V
$$

$$
P_{1} = \left(\begin{array}{cccccccccccccccc} (p_{1} + \mu_{H}) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta_{DH}b_{DV}Y_{14}}{N_{H}} & -g_{1} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \gamma_{D} - g_{2} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \theta_{D}(1-P_{D}) - g_{3} & \phi_{D} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & r_{D} & 0 & -\mu_{H} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & r_{D} & 0 & -\mu_{H} & 0 & 0 & 0 & 0 \\ \frac{\beta_{DH}b_{CV}Y_{17}}{N_{H}} & 0 & 0 & 0 & 0 & 0 & -g_{5} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & r_{C} - g_{6} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \end{array}\right)
$$

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	0	0		$0 - \beta_{DH} b_{DV}$ 0			$0 - \beta_{CH} b_{CV}$	
	0	0	$\boldsymbol{0}$	$\beta_{\scriptscriptstyle DH} b_{\scriptscriptstyle DV}$	$\bf{0}$	0	$\overline{0}$	
	O	0	θ	$\overline{0}$	0	0	0	
	O	0	θ	$\boldsymbol{0}$	0	0	0	
	0	0	θ	θ	0	0	0	
	0	0	θ	θ	0	$\overline{0}$	0	
	0	0	$\overline{0}$	0	0	$\boldsymbol{0}$	$\beta_{CH}b_{CV}$	
	O	θ	Ω	0	Ω	Ω	$\overline{0}$	
$P_2 =$	$\mathbf{\Omega}$	0	θ	0	Ω		0	
	0	0	$\overline{0}$	0	0	0	0	
	$-\mu_{H}$	0	$\boldsymbol{0}$	$\boldsymbol{0}$	Ω	0	0	
	$\boldsymbol{0}$	$-(P_2 + \mu_V)$ 0		$\boldsymbol{0}$	0		0	
	$\overline{0}$	P_{2}	$-g9$	$\boldsymbol{0}$	Ω		0	
	$\overline{0}$	$\boldsymbol{0}$		$\gamma_D - \mu_V$	$\boldsymbol{0}$	0	O	
	0	$\boldsymbol{0}$	$\boldsymbol{0}$		$0 - (P_3 + \mu_V)$	$\overline{0}$	0	
	0	θ	$\boldsymbol{0}$	$\boldsymbol{0}$	P_{3}	$- g_{10}$	0	
		0	0	$\mathbf{0}$	0	γ_c	$-\mu_{\scriptscriptstyle V}$	

Now, using the fact that $G \ge 0$ and that the matrix $P(Y)$ is quasi-positive, it follows that (21) is positively-invariant in R^{17}_{+} . Thus, we have established positivity for all the state variables in model (20) for all time. Next, we claim the following:

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Theorem 2 Let

$$
{}^{'}S_{H}(t), E_{D}(t), I_{D1}(t), I_{D2}(t), I_{WD}(t), R_{D}(t), E_{C}(t), I_{C1}(t), I_{C2}(t), I_{WC}(t), R_{C}(t),
$$

$$
I_{MD}(t), S_{MC}(t), E_{MC}(t), I_{MC}(t)
$$

Be trajectories of the system (20) with initial condition and the biological feasible region given by the set $D = D_a \times D_b \times D_c \subset R_+^{12} \times R_+^2 \times R_+^3 \subset R_+^{17}$ where
 $D_a = \{ (S_H, E_D, I_{D1}, I_{D2}, I_{WD}, R_D, S_H E_C, I_{C1}, I_{C2}, I_{WC}, R_C) \in R_+^{12} : N_H \le \frac{\Lambda_H}{\Lambda_H} \}$

$$
D_a = \{ (S_H, E_D, I_{D1}, I_{D2}, I_{WD}, R_D, S_H, E_C, I_{C1}, I_{C2}, I_{WC}, R_C) \in R_+^{12} : N_H \le \frac{\Lambda_H}{\mu_H} \},
$$

\n
$$
D_b = \{ (S_{MD}, E_{MD}, I_{MD}) \in R_+^3 : N_S \le \frac{\Lambda_{VD}}{\mu_V} \},
$$

\n
$$
D_b = \{ (S_{MD}, E_{MD}, I_{MD}) \in R_+^3 : N_S \le \frac{\Lambda_{VD}}{\mu_V} \},
$$

Is positively-invariant and attracts all the positive trajectories of the model (20)

 Proof: Adding up the right hand side of the vector field for the human population in (20) yields filian projects of the *dN*
dN
dt

EXECUTE: By
Proot: Adding up the right hand side of the vector field for the human population in (20
yields

$$
\frac{dN_H}{dt} = \Lambda_{VD} - \mu_H (S_H + E_D + I_{D1} + I_{D2} + I_{WD} + R_D + E_C + I_{C1} + I_{C2} + I_{WC} + R_C) - \alpha_D \delta_D I_{D2} - \delta_D I_{WD} - \alpha_C \delta_C I_{C2} - \delta_C I_{WC}
$$

$$
\leq \Lambda_H - \mu_H N_H
$$
(23)

Similarly

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\n
$$
\frac{dN_{VD}}{dt} = \Lambda_{VD} - \mu_V (S_{MD} + E_{MD} + I_{MD})
$$
\n
$$
= \Lambda_{VD} - \mu_V N_{VD}
$$
\n
$$
\frac{dN_{VC}}{dt} = \Lambda_{VC} - \mu_V (S_{MC} + E_{MC} + I_{MC})
$$
\n
$$
= \Lambda_{VC} - \mu_V N_{VC}
$$
\n
$$
= \Lambda_{VC} - \mu_V N_{VC}
$$
\n(25)
\nFurthermore, since the hand right side of the above equality is bounded, it follows that by the comparison theorem [18], the solutions of (20) can be given as
\n
$$
\frac{dN_H}{dt} = \Lambda_{VD}(0) \exp(-\mu_V(t) + (S_H + E_D + I_{D1} + I_{D2} + I_{WD} + R_D + E_C + I_{C1} + I_{C2} + I_{WC} + R_C) - \alpha_D \delta_D I_{D2} - \delta_D I_{WD} - \alpha_C \delta_C I_{C2} - \delta_D I_{CD}
$$

Furthermore, since the hand right side of the above equality is bounded, it follows that by the comparison theorem [18], the solutions of (20) can be given as

=
$$
\Lambda_{VC} - \mu_V N_{VC}
$$
 (25)
Furthermore, since the hand right side of the above equality is bounded, it follows that by the
comparison theorem [18], the solutions of (20) can be given as

$$
\frac{dN_H}{dt} = \Lambda_{VD}(0) \exp(-\mu_V(t) + (S_H + E_D + I_{D1} + I_{D2} + I_{WD} + R_D + E_C + I_{C1} + I_{C2} + I_{WC} + R_C) - \alpha_D \delta_D I_{D2} - \delta_D I_{WD} - \alpha_C \delta_C I_{C2} - \delta_C I_{WC}
$$

 $\leq \Lambda_H - \mu_H N_H$ (26)

$$
\leq \Lambda_H - \mu_H N_H
$$

\n
$$
N_h(t) \frac{dN_H}{dt} \leq N_{VD}(0) \exp(-\mu_V(t) + \frac{\Lambda_{VD}}{\mu_V} [1 - \exp(-\mu_V(t))], \text{ so that}
$$

\n
$$
Lim_{t\to\infty} \sup N_H(t) \leq \frac{\Lambda_{VD}}{\mu_V},
$$

$$
N_{\text{VC}}(t) = N_{\text{VC}}(0) \exp(-\mu_{\text{V}}(t) + \frac{\Lambda_{\text{VC}}}{\mu_{\text{V}}}[1 - \exp(-\mu_{\text{V}}(t))], \text{ so that}
$$

$$
\lim_{t \to \infty} \sup N_{\text{VCV}}(t) \le \frac{\Lambda_{\text{VC}}}{\mu_{\text{V}}}.
$$

 $\mu_{_V}$

 $\mu_{_V}$

In particular, if

$$
N_{\rm H} \le \frac{\Lambda_{\rm H}}{\mu_{\rm H}}, \text{ then } N_{\rm H}(t) \le \frac{\Lambda_{\rm H}}{\mu_{\rm H}} \text{ for all } t > 0, \text{ then } N_{\rm VD}(0) \le \frac{\Lambda_{\rm VD}}{\mu_{\rm V}}, \quad N_{\rm VD}(t) \le \frac{\Lambda_{\rm VD}}{\mu_{\rm V}}
$$

to 0 and $N_{\rm VC}(0) \le \frac{\Lambda_{\rm VC}}{\mu_{\rm V}}, \text{ then } N_{\rm VC}(t) \le \frac{\Lambda_{\rm VC}}{\mu_{\rm V}} \text{ for all } t > 0$

For all

Hence, the closed set D is positively invariant under the flow of the system (20). As a result, D is an attractor and no trajectory goes out of any boundary of D.

4.0 Local Asymptotic stability (LAS) of Disease-free Equilibrium (DFE)

The model system (4) has a DFE given by,

$$
\xi_0 = (S_H^0, E_D^0, I_{D1}^0, I_{D2}^0, I_{WD}^0, R_D^0, E_C^0, I_{C1}^0, I_{WC}^0, R_C^0, S_{MD}^0, E_{MD}^0, I_{MD}^0, S_{MC}^0, E_{MC}^0, I_{MC}^0)
$$

=
$$
(\frac{\Lambda_H}{\mu_H}, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, \frac{\Lambda_{VD}}{\mu_V}, 0, 0, \frac{\Lambda_{VC}}{\mu_V}, 0, 0).
$$
 (27)

The method of next generation matrix operator method by [19] is used to investigated the locally asymptotically stable (LAS) of the diseases-free equilibrium (DFE). Using the notations S and T to represent the one used in $[19]$, where the matrices S is the new infection terms and T is the transfer terms. Here

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$$
S = (S_{1(12x5)} \t S_{2(12x7)}) \t and \t T = (T_{(12x12)}) \t (28)
$$

$$
S = (S_{1(12x5)} \quad S_{2(12x7)}) \quad and \quad T = (T_{(12x12)}) \tag{28}
$$
\n
$$
= \begin{pmatrix}\n0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
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0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 &
$$

$$
S_2 = \left(\begin{array}{cccccccc} 0 & 0 & 0 & 0 & 0 & \beta_{DH} b_{DV} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \beta_{CH} b_{CV} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \beta_{CH} b_{CV} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \beta_{CV} b_{CV} \eta_{CI} \frac{S_{MC}}{N_H} & \beta_{CV} b_{CV} \eta_{C2} \frac{S_{MC}}{N_H} & \beta_{CV} b_{CV} \eta_{CS} \frac{S_{MC}}{N_H} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{array}\right)
$$

and

$$
T = \left(\begin{array}{cccccccccccccccc} g_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ -\gamma_D & g_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -\theta_D(1-P_D) & g_3 & -\phi_D & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \theta_D P_D & g_4 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & g_5 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\gamma_C & g_6 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\theta_C(1-P_C) - g_7 & -\phi_C & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\theta_C P_C & 0 & g_8 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & g_9 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & g_{10} & \mu_V & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & g_{10} & \mu_V & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\gamma_{MC} & \mu_V \end{array}\right)
$$

 $\overline{}$ $\overline{}$

)

J

The effective reproduction number of the model (20) $\mathfrak{R}_0 = \rho(ST^{-1}) = \max[\mathfrak{R}_{0D}, \mathfrak{R}_{0C}],$

With ρ being the spectral radius of the largest Eigen value associated with matrix ST^{-1} , is given by

$$
\mathfrak{R}_{0D} = \sqrt{\frac{\beta_{DH}\beta_{DV}b_{DV}^2\Lambda_{VD}\mu_H\gamma_{MD}(g_3g_4(\gamma_D + g_2\eta_{D1}) + g_4\gamma_D\eta_{D2}\theta_D(1 - P_D) + P_D\gamma_D\theta_D(g_3\eta_{D3} + \eta_{D2}\phi_D)}{\Lambda_H\mu_V^2g_1g_2g_3g_4g_9}},
$$
\n
$$
\mathfrak{R}_{02} = \sqrt{\frac{\beta_{CH}\beta_{CV}b_{CV}^2\Lambda_{VC}\mu_H\gamma_{MC}(g_7g_9(\gamma_C + g_6\eta_{C1}) + g_8\gamma_C\eta_{C2}\theta_C(1 - P_C) + P_C\gamma_C\theta_C(g_7\eta_{C3} + \eta_{C2}\phi_C)}{\Lambda_H\mu_V^2g_5g_6g_7g_8g_{10}}},
$$
\n(30)

Where, $g_1 = \gamma_D + \mu_H g_2 = \delta_D + \mu_H + \theta_D g_3 = \alpha_D \delta_D + \tau_D + \mu_H g_4 = \delta_D + \phi_C + \mu_H g_5 = \gamma_{MC} + \mu_H$ $\mathcal{L}_{\mathcal{L}} g_{6} = \delta_{C} + \mu_{H} + \theta_{C} g_{7} = \alpha_{C} \delta_{C} + \tau_{C} + \mu_{H} g_{8} = \delta_{C} + \phi_{C} + \mu_{H} g_{9} = \gamma_{MD} + \mu_{V} g_{10} = \gamma_{MC} + \mu_{V}$

Using Theorem 2 in [19] we claim the following result:

The diseases free state (DFE) of the system (20), is locally asymptotically stable (LAS) in D if \mathcal{R}_{DC} < 1 and unstable if \mathcal{R}_{DC} > 1.

5.0 Conclusion

The threshold quantity $\mathfrak{R}_{\scriptscriptstyle{DC}}$, is the effective reproduction number of the disease. It is the average number of secondary dengue infections generated by infected individual in a completely susceptible population. The epidemiological implication of reproduction number is that Dengue and Chikungunya can be eradicated (asymptotically) from a population where there are instances

of misdiagnosed of either disease, when \Re_{DC} < 1, if the initial sizes of the sub-populations of the model (4) lie in the basin of attraction of the DFE. This implies that if a small number of infectious Dengue and Chikungunya patients enters such a population where there are cases of misdiagnosis, it will not lead to a large Dengue or Chikungunya outbreak in the population.

References

- [1] Costa-da-silva, A. L., Ioshino, R.S., Correa de Araujo, H. R., Kojin B.B., Zanotto,P.M., Oliveira, D.B., Melo, S.R., Durigon, E.L. and Capurro, M.L. (2017). Laboratory strains of Aedes aegypti are competent to Brazilian Zikavirus. Plos ONE 12(2):eo171951 doi:10.137/Journal.Pone.0171951
- [2] Centers for Disease Control and prevention (2018)[. www.cdc.gov/Chikungunya/index.](http://www.cdc.gov/Chikungunya/index) Html.
- [3] Burt F.J., Chen W., Miner J.J., Lenschow D.J., Merits A., Schnettler E., Kohl A., Rudd P.A., Taylor A, Herrero L.J., Zaid A.,Ng LFP, Mahalingan S. (2017) Chikungunya virus: an update on the biology and pathogenesis of the emerging pathogen. Lancet Infect Dis. Apr; 17 (4) :e107-e117.doi:10.1016/s1473-3099 (16)30885-1.
- [4] Sergon, K., Njuguna, C., and Kalani, R. (2008). Seroprevalence of chikungunya virus (CHIKV) infection on Lamu Island, Kenya, October 2004. Am J Trop Med Hyg; 78:33{37. Google Scholar PubMed.
- [5] World Health Organization (W.H.O) (2006): Outbreak news on chikungunya and dengue, south-west Indian Ocean. Wkly Epidemiol Rec; 81:1068
- [6] Herve Zeller, Wimvan Bortel, and Bertrand Sudre (2016). Chikungunya: it's History in Africa andAsia and its speedto New Regions in 2013-2014. The Journalof Infectious Disease, vol. 214, pages S436-S440.
- [7] S.V. Mayer, R.B. Tesh, N. Vasilakis (2017). The emergence of arthropod borne viral diseases: A global prospective on dengue, chikungunya and Zika fever.

Akhaze and Olowu O.O. / NIPES Journal of Science and Technology Research 3(4) 2021 pp. 1-15

- [8] Dejinirahisai, Noisakran, S., Onloimoon, N., Songprakhan, P., Hsiao, H., Chokephaibulkit, K. and Perng, G.C. (2010). Cells in Dengue virus infection in vivo, Advance in virology, (2010), Article ID 164878, 15 pages.
- [9] World Health Organization (2011). Dengue hemorahagic fever w.w.w. who. Int/mediacentre/ factsheets/fs 117lenl.
- [10] Seed, C.R., Kiely P., Hyland, C.A. and Keller, A.J. (2009) The risk of dengue transmission by blood during a 2004 outbreak in Cairns, Australia. Transfusion (Paris) 49: 1482{1487.
- [11]Schmid, M.A., Diamond, M.S. and Harris, E. (2014). Dendritic cells in dengue virus infection: targets of virus replication and mediators of immunity. https://doi.org 10.3389.
- [12]A. Agarwal, P. k. Dash, A. K. Singh, S. Sharma, N. Gopalan, P. V. Lakshmana Rao, M. M Parda, Paul Reiter (2014). Evidence of Experimental Vertical Transmission of Emerging Novel ECSA Genotype of Chikungunya Virus in Aedes aegypti. Journals.plos Org/Plosntds/article? Id=10.1371/Journal.pntd.0002990.
- [13]Faneye A, Idika, N. Motayo, B. O., Adesanmi, A. and Afocha, E. (2013). Serological evidence of recent dengue virus infection among febrile children in a semi arid zone. Am. J. Infect. Dis., 9: 10.
- [14] Pan American Health organization (2016). Health Emergencies program. [https://www.paho,](https://www.paho/) Org/ hq / index. Php? Option=Com_ topic.
- [15]C. M. Yuen, F. Amanullah, A. Dharmadhikari, E.A. Nardell, J. A Seddon, I. Vasilyera, Y. Zhao, S. Kesharjee, M. C. Becerra (2015). Turning off the tap: Stopping tuberculosis transmission through active case-finding and prompt effective treatment. Vol.386, pages 2334-2343.
- [16]Y. Dumont and F. Chiroleu, (2010). Vector control for the Chikungunya disease. Math. Biosci. Eng. 7, pp. 313-345.
- [17] O.O. Okuneye and A.B. Gumel, (2016). Analysis of a Temperature and Rainfall Dependent Model for Malaria Transmission Dynamics}, Mathematical Biosciences. DOI 10.1016/j.mbs.2016.03.013, 2015.
- [18]V. Lakshmikantham, (1991). Stability analysis of nonlinear systems, {SIAM Review} 33(1): 152- 154.
- [19]Van den Driessche and watmough, J. (2002). 'Reproduction numbers and sub-theshold endemic equilibria for compartmental models of disease transmission mathematical Biosciences vol.180.