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Modelling the super-infection of two strains of dengue virus

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Abstract

Dengue is one of the vector borne diseases that threatened human race. It is imperative to understand the transmission dynamics of dengue, so that proficient and useful control can be developed. In this paper, we formulated dynamic transmission of two strains super-infection dengue. We used next generation matrix to obtain the basic reproduction numbers $\mathcal{R}_1$, $\mathcal{R}_2$, $\mathcal{R}_{12}$. The obtained basic reproduction numbers are then used to test for stabilities whenever $\mathcal{R}_1 < 1$, $\mathcal{R}_2 < 1$, $\mathcal{R}_{12} < 1$, disease free-equilibrium is globally asymptotically stable or otherwise unstable. We also carried out numerical simulation with the aid of python software. Our results reveal that decreasing the value of tansmission probability α, slows down the spread of the disease.

Keywords: Dengue, Super-infection, Modelling, Analysis, Dynamics, Transmission

Introduction

Dengue is an infectious disease and caused by tiny pathogenic destructive organisms and can be transmittable upon contact with an infected person by biting. It is commonly found in tropical and subtropical areas around the world. The reproduction of the vectors that carry and transmit the disease are enhanced by warm weather and rain. Mosquitoes are multiplied and replicated on a stand still water, it is best to note that human is their source of nutrient. In Cairo, Egypt and Batavia, Indonesia in 1779 was the first time the cases of dengue fever were reported. 50,000 cases were reported in 1818 when dengue pandemics occurred. Several cases were reported not until 1944 when a breakthrough in treatment came through Dr Albert Sabin. Dengue is the fast-spreading vector-borne viral disease that transmitted to human by the bite of an infected mosquito. The predominant vector for dengue infection is named aedes aegypti mosquito while other species can also transmit the dengue with different degree of capability. Dengue virus causes a severe health issues in tropical and subtropical across the globe and Dengue viruses are major contributors to illness and death worldwide. The rising global spread of dengue virus and the inadequacy of an approved vaccine or anti-viral has incited rigorous research during the past few years. Several researchers have carried out investigations on the dynamic of dengue virus using mathematical and statistical models. Abidemi et al [1] considered two-strain compartmental dengue model with flexible humans and mosquitoes populations sizes. Their model included two control measures to predict the transmission and effective control strategy for

dengue in Madeira Island. They got basic reproduction ration associated with the model and established local and global asymptotic stability associated with the model. They concluded by their numerical results that a strategy which is based on Dengvaxia vaccine and adulticide is the most effective strategy for controlling dengue disease transmission in their considered scenarios. Ndelamo et al. [2] examined the effect of treatment of Dengue fever disease. They suggested a nonlinear mathematical model and analysed quantitatively using the stability theory of the differential equations. They concluded that that the disease-free equilibrium is locally and globally asymptotically stable if the reproduction number is less than one. They simulated the model numerically in order to look into the sensitivity of varied parameter responsible for dengue fever with treatment. Hamdan and Kilicman [3] used a deterministic mathematical model by considering the effect of temperature to examine o the dengue dynamics. Their model shew possibility of backward bifurcation and they observed oscillatory behaviour in their solution via numerical simulation. Manore et al. [4] sought to understand the differences in transient and endemic behavior of chikungunya and dengue. They adopted mathematical model for mosquito-borne disease and derives analytical threshold, dimensionless parameters for transmission and performed some analysis on the obtained models. They concluded that chikungunya and dengue demonstrate different vagrant dynamics and long-run endemic levels. Esteva and Vargas [5] explored mathematical model for transmission of dengue fever in a constant human population and variable vector population. They carried out global analysis and then established the global stability of the endemic equilibrium. Adak and Jana [6] created a mathematical model using type-2 fuzzy inference system to foretell appropriate circumstances for dengue eruption so that control measures can be executed as soon as possible. For proper understanding of the system, they used MATLAB software to generate various simulation works. Side and Noorani [7] used system of differential equations to model the population dynamics of vector transmission of dengue fever by looking at re-breeding value based on the number of reported cases of dengue fever in South Sulawesi, Indonesia and Selangor, Malaysia. They were able to establish by the models that both countries arrived at maximum levels in short period of time. In simulation of their models, they were able to indicate that dengue fever has not become endemic in either country. Gubler et.al [8] was first to report the case of concurrent human infection with two dengue viruses.

The nonlinear mathematical model to describe the transmission dynamic and control of dengue disease was discussed by [9] within the context of the interplay between human and mosquitoS population. They obtained the dengue-free equilibrium for the model and showed the globally asymptotically stability. Their simulations examined how Wolbachia coverage and the fraction of symptomatic infecting persons who would develop severe dengue affect community dengue dynamics. Their findings show that Wolbachia control may help reduce dengue transmission in the community, as well as early hospitalization for symptomatic infected persons with moderate clinical symptoms to speed recovery. [10] presented a compartmental deterministic model including human prevention and vector control interventions for the dynamics of dengue fever spread. The effective reproduction number was obtained and presented the local stability analysis for the models The performed numerical simulations of different strategies of control combination and found that dengue prevalence can be reduced in a community by implementing any control intervention which combines human prevention and vector control measures.

Peijiang et. al [11] carried out investigation on stochastic disturbances involving both human and mosquito populations according to the characteristics of the diseases.They used suitable Lyapunov function, the uniqueness and existence of positive solution to the stochastic differential equation model and also investigated exponential stability of the problem. Asamoah et al [12] analysed an optimal control model of dengue infection with partial immune and asymptomatic individuals. They introduced four time-dependent control measures, and concluded that the equilibrium points are globally asymptotically stable using Lyapunov functions, Several other works on dengue can be found in [13–16].

The aim of this paper is to use mathematical modeling to investigate the epidemiological influence of super-infection on the two strains of the dengue transmission dynamics in a population. A proper understanding of dengue vector-human interactions would improve knowledge of the pathogen transmission and could expose aims for reducing risk and cracking pathogen communication sequences. The rest of this paper is organized as follows. In section 2, we present the dynamic models for the two strains Dengue epidemics, in which the parameters and system of ordinary differential equations describing the problem are defined. In section 3, the mathematical analysis for the formulated problem are carried out. Section 4, the numerical simulation to the problem are presented while section 5 give the concluding remarks.

Methods

Mathematical formulation of the model

The human population is divided into 5 classes namely, the susceptible class H_s, those infected with strain 1 virus H_{I_1}, those infected with strain 2 virus H_{I_2}, those that are super-infected with strain-1 and strain-2 viruses $H_{I_{12}}$, and those that have recovered from all the dengue virus infection, H_R. Similarly, in the mosquito population, there are 4 classes, namely, the susceptible class M_s, those infected with strain 1 virus M_{I_1}, those infected with strain 2 virus M_{I_2}, those that are super-infected with strain-1 and strain-2 viruses $M_{I_{12}}$. Hence, we can write the total population, H_N, of human, and total population, M_N of mosquitoes as $H_N = H_s + H_{I_1} + H_{I_2} + H_{I_{12}} + H_R$ and $H_N = M_s + M_{I_1} + H_{I_2} + M_{I_{12}}$, respectively. In the human population, it is assumed that humans are recruited into the susceptible class at the rate, π_H. Humans in the susceptible class become infected with strain-1 virus or strain-2 virus when bitten by mosquitoes in the classes, $M_{I_1} or M_{I_2}$, respectively. The biting rate of mosquitoes carrying strain-1 virus is β_1, while the biting rate of mosquitoes carrying strain-2 virus is β_2. The probability of transmission of any of the dengue strains from infected mosquitoes to humans, and vice versa is put at α. A super-infection of the two dengue virus strains in the human and mosquito populations occurs when a mosquito in the class, M_{I_1} successfully bites a human in the class, H_{I_2}, and when a mosquito in the class, M_{I_2} successfully bites a human in the class, H_{I_1}. The biting rate in this case is β_{12}. It is assumed that humans infected with strain-1 virus can recover at the rate γ_1, while the rate of recovery for those that are super-infected with the two srain is γ_2. Those who recover from the dengue infection cannot be reinfected with the virus. In addition to human natural mortality rate, μ_0, the classes H_{I_1} and H_{I_2} suffers the same dengue-induced death rate, μ_1. However, the death rate, μ_2, due to super-infection is higher than μ_1. Similarly, in the mosquito population, mosquitoes are recruited into the susceptible class at the

rate π_M, the mosquitoes become infected with strain-1 or strain-2 virus when they bite humans in the classes, $H_{I_1} or H_{I_2}$, respectively. The natural mortality rate for mosquitoes is given at μ_m. From the flow diagram (Fig. 1) and the model description given above, we arrive at the following system of non-linear ordinary differential equations which models the super-infection of dengue virus in human and aedes aegypti populations;

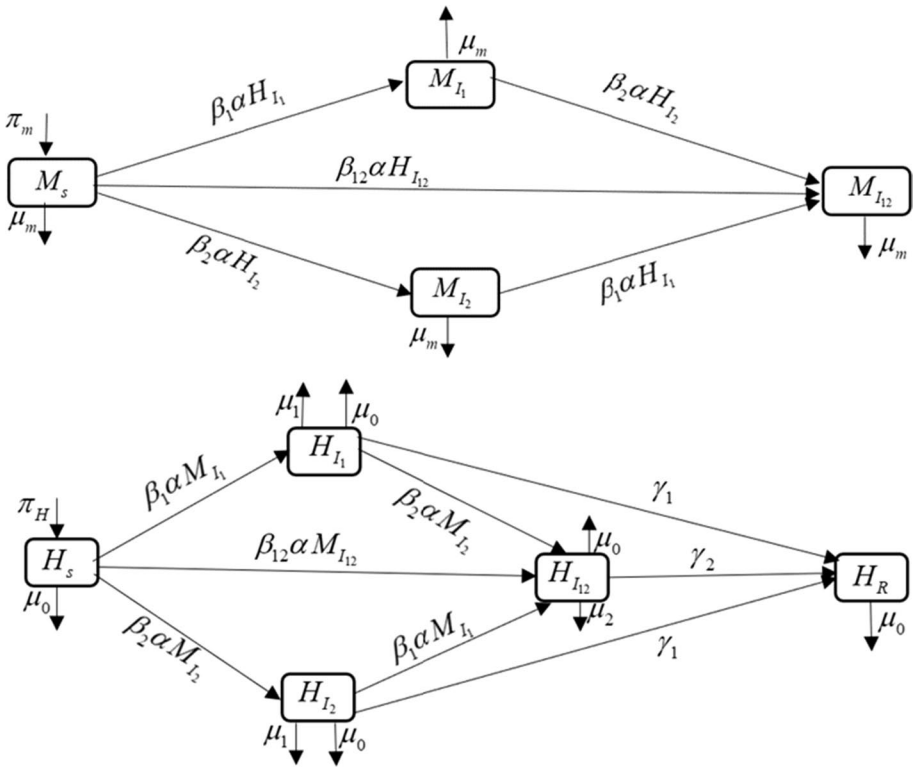

Fig. 1 Schematic diagram depicting the dengue transmission dynamics

$$
\begin{aligned}
\frac{\mathrm{d}M_s}{\mathrm{d}t} &= \pi_m - \beta_1\alpha H_{I_1}M_s - \beta_2\alpha H_{I_2}M_s - \beta_{12}\alpha H_{I_{12}}M_s - \mu_m M_s,\\
\frac{\mathrm{d}MI_1}{dt} &= \beta_1\alpha H_{I_1}M_s - \beta_2\alpha H_{I_2}M_{I_1} - \mu_m M_{I_1},\\
\frac{\mathrm{d}M_{I_2}}{\mathrm{d}t} &= \beta_2\alpha H_{I_2}M_s - \beta_1\alpha H_{I_1}M_{I_2} - \mu_m M_{I_2},\\
\frac{\mathrm{d}M_{I_{12}}}{\mathrm{d}t} &= \beta_1\alpha H_{I_1}M_{I_2} + \beta_2\alpha H_{I_2}M_{I_1} + \beta_{12}\alpha H_{I_{12}}M_s - \mu_m M_{I_{12}},\\
\frac{\mathrm{d}H_s}{\mathrm{d}t} &= \pi_H - \beta_1\alpha M_{I_1}H_s - \beta_2\alpha M_{I_2}H_s - \beta_{12}\alpha M_{I_{12}}H_s - \mu_0 H_s,\\
\frac{\mathrm{d}H_{I_1}}{\mathrm{d}t} &= \beta_1\alpha M_{I_1}H_s - \beta_2\alpha M_{I_2}H_{I_1} - (\mu_0+\mu_1+\gamma_1)H_{I_1},\\
\frac{\mathrm{d}H_{I_2}}{\mathrm{d}t} &= \beta_2\alpha M_{I_2}H_s - \beta_1\alpha M_{I_1}H_{I_2} - (\mu_0+\mu_1+\gamma_1)H_{I_2},\\
\frac{\mathrm{d}H_{I_{12}}}{\mathrm{d}t} &= \beta_1\alpha M_{I_1}H_{I_2} + \beta_2\alpha M_{I_2}H_{I_1} + \beta_{12}\alpha M_{I_{12}}H_s - (\mu_0+\mu_2+\gamma_2)H_{I_{12}},\\
\frac{\mathrm{d}H_R}{\mathrm{d}t} &= (H_{I_1}+H_{I_2})\gamma_1 + H_{I_{12}}\gamma_2 - \mu_0 H_R.
\end{aligned}
\tag{1}
$$

State variables	Description
H_S	Humans that are susceptible to any of the strains of dengue viruses or both
H_{I_1}	Humans that are infected with strain-1 dengue virus only
H_{I_2}	Humans that are infected with strain-2 dengue virus only
$H_{I_{12}}$	Humans that are co-infected with strain-1 and strain-2 dengue viruses
H_{R_1}	Humans that have recovered from strain-1 infection
H_{R_2}	Humans that have recovered from strain-1 infection
H_R	Humans that have recovered from the super-infection
M_S	Recruitment rate of mosquitoes into susceptible class
M_{I_1}	Mosquitoes that are infected with strain-1 dengue virus only
M_{I_2}	Mosquitoes that are infected with strain-2 dengue virus only
$M_{I_{12}}$	Mosquitoes that are co-infected with strain-1 and strain-2 dengue viruses

Mathematical analysis

Disease-free equilibrium and basic reproduction number of the model

The system of nonlinear above equations has disease-free equilibrium

$$\Xi_0 = \left(\frac{\pi_m}{\mu_m}, 0, 0, 0, \frac{\pi_H}{\mu_0}, 0, 0, 0, 0\right),$$

which is always practicable. This is the solution to the right-hand side of (1) when when set to zero, in the absence of disease infections in the population.

The basic reproduction number

With Watmough and Driessche [19], we use next-generation method to determine the basic reproduction number.

$$\mathcal{F} = \begin{bmatrix} \beta_1 \alpha H_{I_1} M_s \\ \beta_2 \alpha H_{I_2} M_s \\ \beta_1 \alpha H_{I_1} M_{I_2} + \beta_2 \alpha H_{I_2} M_{I_1} + \beta_{12} \alpha H_{I_{12}} M_s \\ \beta_1 \alpha M_{I_1} H_s \\ \beta_2 \alpha M_{I_2} H_s \\ \beta_1 \alpha M_{I_1} H_{I_2} + \beta_2 \alpha M_{I_2} H_{I_2} + \beta_{12} \alpha M_{I_{12}} H_s \end{bmatrix}$$

and

$$\mathcal{V} = \begin{bmatrix} \beta_2 \alpha H_{I_2} M_{I_1} + \mu_m M_{I_1} \\ \beta_1 \alpha H_{I_1} M_{I_2} + \mu_m M_{I_2} \\ \mu_m M_{I_{12}} \\ \beta_2 \alpha M_{I_2} H_{I_1} + \left(\mu_0 + \mu_1 + \gamma_1\right) H_{I_1} \\ \beta_1 \alpha M_{I_2} H_{I_2} + \left(\mu_0 + \mu_1 + \gamma_1\right) H_{I_2} \\ \left(\mu_0 + \mu_2 + \gamma_2\right) H_{I_{12}} \end{bmatrix}.$$

Therefore

$$F=\begin{pmatrix} 0 & 0 & 0 & \beta_1\alpha M_s & 0 & 0 \\ 0 & 0 & 0 & 0 & \beta_2\alpha M_s & 0 \\ \beta_2\alpha H_{I_2} & \beta_1\alpha H_{I_1} & 0 & \beta_1\alpha M_{I_2} & \beta_2\alpha M_{I_1} & \beta_{12}\alpha M_s \\ \beta_1\alpha H_s & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_2\alpha H_s & 0 & 0 & 0 & 0 \\ \beta_1\alpha H_{I_2} & \beta_2\alpha H_{I_1} & \beta_{12}\alpha H_s & \beta_1\alpha M_{I_2} & \beta_2\alpha M_{I_1} & 0 \end{pmatrix},$$

$$V=\begin{pmatrix} \beta_2\alpha H_{I_2}+\mu_m & 0 & 0 & 0 & \beta_2\alpha M_{I_1} & 0 \\ 0 & \beta_1\alpha H_{I_1}+\mu_m & 0 & \beta_1\alpha M_{I_2} & 0 & 0 \\ 0 & 0 & \mu_m & 0 & 0 & 0 \\ 0 & \beta_2\alpha H_{I_1} & 0 & \beta_2\alpha M_{I_2}+(\mu_0+\mu_1+\gamma_1) & 0 & 0 \\ 0 & \beta_1\alpha H_{I_2} & 0 & 0 & \beta_1\alpha M_{I_2}+(\mu_0+\mu_1+\gamma_1) & 0 \\ 0 & 0 & 0 & 0 & 0 & \mu_0+\mu_2+\gamma_2 \end{pmatrix}.$$

At DFE

$$F=\begin{pmatrix} 0 & 0 & 0 & \beta_1\alpha\frac{\pi_m}{\mu_m} & 0 & 0 \\ 0 & 0 & 0 & 0 & \beta_2\alpha\frac{\pi_m}{\mu_m} & 0 \\ 0 & 0 & 0 & 0 & 0 & \beta_1\alpha\frac{\pi_m}{\mu_m} \\ \beta_1\alpha\frac{\pi_H}{\mu_0} & 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_2\alpha\frac{\pi_H}{\mu_0} & 0 & 0 & 0 & 0 \\ 0 & 0 & \beta_{12}\alpha\frac{\pi_H}{\mu_0} & 0 & 0 & 0 \end{pmatrix},$$

$$V=\begin{pmatrix} \mu_m & 0 & 0 & 0 & 0 & 0 \\ 0 & \mu_m & 0 & 0 & 0 & 0 \\ 0 & 0 & \mu_m & 0 & 0 & 0 \\ 0 & 0 & 0 & \mu_0+\mu_1+\gamma_1 & 0 & 0 \\ 0 & 0 & 0 & 0 & \mu_0+\mu_1+\gamma_1 & 0 \\ 0 & 0 & 0 & 0 & 0 & \mu_0+\mu_2+\gamma_2 \end{pmatrix},$$

$$V^{-1}=\begin{bmatrix} \frac{1}{\mu_m} & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{1}{\mu_m} & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{1}{\mu_m} & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{1}{\mu_0+\mu_1+\gamma_1} & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{1}{\mu_0+\mu_1+\gamma_1} & 0 \\ 0 & 0 & 0 & 0 & 0 & \frac{1}{\mu_0+\mu_2+\gamma_2} \end{bmatrix},$$

$$FV^{-1}=\begin{bmatrix} 0 & 0 & 0 & \frac{\beta_1\alpha\pi_m}{\mu_m(\mu_0+\mu_1+\gamma_1)} & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{\beta_2\alpha\pi_m}{\mu_m(\mu_0+\mu_1+\gamma_1)} & 0 \\ 0 & 0 & 0 & 0 & 0 & \frac{\beta_1\alpha\pi_m}{\mu_m(\mu_0+\mu_2+\gamma_2)} \\ \frac{\beta_1\alpha\pi_H}{\mu_0\mu_m} & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_2\alpha\pi_H}{\mu_m\mu_0} & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_{12}\alpha\pi_H}{\mu_m\mu_0} & 0 & 0 & 0 \end{bmatrix}.$$

The eigenvalues of FV^{-1} are $\pm\frac{\alpha\beta_1}{\mu_m}\sqrt{\frac{\pi_H\pi_m}{\mu_0(\mu_0+\mu_1+\gamma_1)}}$, $\pm\frac{\alpha\beta_2}{\mu_m}\sqrt{\frac{\pi_H\pi_m}{\mu_0(\mu_0+\mu_1+\gamma_1)}}$ and $\pm\frac{\alpha\beta_{12}}{\mu_m}\sqrt{\frac{\pi_H\pi_m}{\mu_0(\mu_0+\mu_2+\gamma_2)}}$. Hence, the basic reproduction number of the model is given by

$$\mathcal{R}_0=\max(\mathcal{R}_1,\mathcal{R}_2,\mathcal{R}_{12}),$$

where

$$\mathcal{R}_1 = \frac{\alpha\beta_1}{\mu_m}\sqrt{\frac{\pi_H\pi_m}{\mu_0(\mu_0+\mu_1+\gamma_1)}},\ \mathcal{R}_2 = \frac{\alpha\beta_2}{\mu_m}\sqrt{\frac{\pi_H\pi_m}{\mu_0(\mu_0+\mu_1+\gamma_1)}},$$
$$\mathcal{R}_{12} = \frac{\alpha\beta_{12}}{\mu_m}\sqrt{\frac{\pi_H\pi_m}{\mu_0(\mu_0+\mu_2+\gamma_2)}}.$$

The major difference between the reproduction numbers $\mathcal{R}_1$, $\mathcal{R}_2$ and $\mathcal{R}_{12}$ is the biting rate of the mosquitoes. This shows that what determines the virus strain that dominates in the population is the biting rate of the mosquito carrying a particular virus strain.

Local asymptotic stability of disease-free equilibrium

The local asymptotic stability of the disease-free equilibrium of the model is determined by the eigenvalues of the Jacobian matrix of the right-hand side of (1) evaluated at the disease-free equilibrium. The disease-free equilibrium is locally asymptotically stable if the all the eigenvalues of the Jacobian matrix are negative, otherwise, the disease-free equilibrium is unstable. The Jacobian matrix of the right-hand side of (1) evaluated at the disease-free equilibrium is

$$J(\Xi_0) = \begin{pmatrix} -\mu_m & 0 & 0 & 0 & 0 & -\beta_1\alpha M_S^* & -\beta_2\alpha M_S^* & -\beta_{12}\alpha M_S^* & 0 \\ 0 & -\mu_m & 0 & 0 & 0 & \beta_1\alpha M_S^* & 0 & 0 & 0 \\ 0 & 0 & -\mu_m & 0 & 0 & 0 & \beta_2\alpha M_S^* & 0 & 0 \\ 0 & 0 & 0 & -\mu_m & 0 & 0 & 0 & \beta_{12}\alpha M_S^* & 0 \\ 0 & -\beta_1\alpha H_S^* & -\beta_2\alpha H_S^* & -\beta_{12}\alpha H_S^* & -\mu_0 & 0 & 0 & 0 & 0 \\ 0 & \beta_1\alpha H_S^* & 0 & 0 & 0 & -\phi_1 & 0 & 0 & 0 \\ 0 & 0 & \beta_2\alpha H_S^* & 0 & 0 & 0 & -\phi_1 & 0 & 0 \\ 0 & 0 & 0 & \beta_{12}\alpha H_S^* & 0 & 0 & 0 & -\phi_2 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \gamma_1 & \gamma_2 & -\mu_1 \end{pmatrix} \tag{2}$$

where $\phi_1 = \mu_0+\mu_1+\gamma_1$ and $\phi_2 = (\mu_0+\mu_2+\gamma_2)$. By deleting the rows and columns containing the eigenvalues μ_m, μ_0 and μ_1, we get the submatrix.

$$J'(\Xi_0) = \begin{pmatrix} -\mu_m & 0 & 0 & \beta_1\alpha M_S^* & 0 & 0 \\ 0 & -\mu_m & 0 & 0 & \beta_2\alpha M_S^* & 0 \\ 0 & 0 & -\mu_m & 0 & 0 & \beta_{12}\alpha M_S^* \\ \beta_1\alpha H_S^* & 0 & 0 & -\phi_1 & 0 & 0 \\ 0 & \beta_2\alpha H_S^* & 0 & 0 & -\phi_1 & 0 \\ 0 & 0 & \beta_{12}\alpha H_S^* & 0 & 0 & -\phi_2 \end{pmatrix}$$

Observe that $J'(\Xi_0) = F - V$, where F and V are matrices from the calculation of the basic reproduction number. It has been proved in [19], that all the eigenvalues of $J'(\Xi_0)$ are all negative if $\rho(FV^{-1}) < 1$, where $\rho(FV^{-1})$ is the basic reproduction number, $\mathcal{R}$. Therefore, Ξ_0 is locally asymptotically stable if $\mathcal{R}_1 < 1, \mathcal{R}_2 < 1$ and $\mathcal{R}_{12} < 1$.

Existence and stability of endemic equilibrium

In the absence of strain-2 dengue virus in the population, we have that $M_s = \frac{\pi_m}{\mu_m+\alpha\beta_1 H_{I_1}}, M_{I_1} = \frac{\alpha\beta_1 H_{I_1} M_s}{\mu_m}, H_s = \frac{\pi_H}{\mu_0+\alpha\beta_1 M_{I_1}}, H_{I_1} = \frac{\beta_1\alpha M_{I_1} H_S}{(\mu_0+\mu_1+\gamma_1)}, H_R = \frac{H_{I_1}}{\mu_0}$, satisfies the system

$$\pi_m - \beta_1\alpha H_{I_1} M_S - \mu_m M_S = 0 \tag{3}$$

$$\beta_1 \alpha H_{I_1} M_S - \mu_m M_{I_1} = 0 \tag{4}$$

$$\pi_H - \beta_1 \alpha M_{I_1} H_S - \mu_0 H_S = 0 \tag{5}$$

$$\beta_1 \alpha M_{I_1} H_S - (\mu_0 + \mu_1 + \gamma_1) H_{I_1} = 0 \tag{6}$$

$$H_{I_1} \gamma_1 - \mu_0 H_R = 0$$

Substuting M_{I_1}, H_{I_1} and H_s into (6) gives

$$\begin{aligned}
&(\mu_m^2 \alpha^2 \beta_1^2 \pi_m \pi_H - \mu_m^4 \mu_0 (\mu_0 + \mu_1 + \gamma_1)) H_{1_1} \\
&\quad + [\mu_m \alpha^3 \beta_1^3 \pi_m \pi_H - \mu_m^3 \mu_0 \alpha \beta_1 (\mu_0 + \mu_1 + \gamma_1) \\
&\quad - \mu_m^2 \alpha^2 \beta_1^2 \pi_m (\mu_0 + \mu_1 + \gamma_1) - \mu_m^2 \mu_0 \alpha \beta_1 (\mu_0 + \mu_1 + \gamma_1))] H_{1_1}^2 \\
&\quad - \mu_m \alpha^2 \beta^2 (\mu_0 + \mu_1 + \gamma_1)(\pi_m + \mu_0) H_{1_1}^3 = 0,
\end{aligned} \tag{7}$$

which simplifies to

$$-\mu_m \alpha^2 \beta^2 (\mu_0 + \mu_1 + \gamma_1)(\pi_m + \mu_0) H_{1_1}^3 + \mu_m^3 \mu_0 \alpha \beta_1 (\mu_0 + \mu_1 + \gamma_1)(\mathcal{R}_1^2 - 1)) H_{1_1}^2 \tag{8}$$

$$-\mu_m^2 \alpha \beta_1 (\alpha \beta_1 \pi_m + \mu_0)(\mu_0 + \mu_1 + \gamma_1) H_{1_1}^2 + \mu_m^4 \mu_0 (\mu_0 + \mu_1 + \gamma_1)(\mathcal{R}_1^2 - 1)) H_{1_1} = 0 \tag{9}$$

The solution $H_{I_1} = 0$, corresponds to the strain-1-free equilibrium, which is locally asymptotically stable when $\mathcal{R}_1 < 1$. The remaining equation has a positive root if $\mathcal{R}_1^2 > 1$, and no positive root when $\mathcal{R}_1^2 < 1$. Therefore, the strain-1 only model has a unique endemic equilibrium when $\mathcal{R}_1^2 > 1$. The existence of one endemic equilibrium when $\mathcal{R}_1 > 1$, rules out the possibility of backward bifurcation in the strain-1 only model. Similar argument can be used to show the existence of one endemic equilibrium in the strain-2 only model when $\mathcal{R}_2 > 1$.

Local stability of endemic equilibrium for strain-1 only model

The Jacobian matrix evaluated at the endemic equilibrium of the strain-1 only model is given by

$$J_1 = \begin{pmatrix}
-(\mu_m + \alpha \beta_1 H_{I_1}^*) & 0 & 0 & -\alpha \beta_1 M_s^* & 0 \\
\alpha \beta_1 H_{I_1}^* & -\mu_m & 0 & \alpha \beta_1 M_s^* & 0 \\
0 & -\alpha \beta_1 H_s^* & -(\mu_0 + \alpha \beta_1 M_{I_1}^*) & 0 & 0 \\
0 & \alpha \beta_1 H_s^* & \alpha \beta_1 M_{I_1}^* & -(\mu_0 + \mu_1 + \gamma_1) & 0 \\
0 & 0 & 0 & \gamma_1 & -\mu_0
\end{pmatrix} \tag{10}$$

The eigenvalues of J_1 are μ_0, and the roots of the characteristic equation

$$\lambda^4 + A_1 \lambda^3 + A_2 \lambda^2 + A_3 \lambda + A_4 = 0, \tag{11}$$

where,

$$
\begin{aligned}
A_1 =&(\mu_m + \alpha\beta_1 H_{I_1}^*) + \mu_m + (\mu_m + \alpha\beta_1 M_{I_1}^*) + (\mu_0 + \mu_1 + \gamma_1),\\
A_2 =&(\mu_m + \alpha\beta_1 H_{I_1}^*)(\mu_m + (\mu_m + \alpha\beta_1 M_{I_1}^*) + (\mu_0 + \mu_1 + \gamma_1)) + \mu_m(\mu_0 + \mu_1 + \gamma_1)(1 - \mathcal{R}_1'^2),\\
A_3 =&\alpha\beta_1 M_s^* \alpha\beta_1 H_s^* (\alpha\beta_1 H_{I_1}^* + \alpha\beta_1 H_s^*) + \mu_m + \alpha\beta_1 H_{I_1}^*)(\mu_m + \alpha\beta_1 M_{I_1}^*)[\mu_m + (\mu_0 + \mu_1 + \gamma_1)]\\
&+ (\mu_m + \alpha\beta_1 H_{I_1}^* + \mu_0 + \alpha\beta_1 M_{I_1}^*)\mu_m(\mu_0 + \mu_1 + \gamma_1)[1 - \mathcal{R}_1'^2],\\
A_4 =&(\mu_m + \alpha\beta_1 H_{I_1}^*)(\mu_m + \alpha\beta_1 M_{I_1}^*)\mu_m(\mu_0 + \mu_1 + \gamma_1)[1 - \mathcal{R}_1'^2)] + \mu_0\alpha^3\beta_1^3 M_s^* H_{I_1}^* H_s^*,
\end{aligned}
$$

where

$$
\mathcal{R}_1'^2 = \frac{\alpha^2\beta_1^2\pi_m\pi_H}{\mu_m^2\mu_0(\mu_0 + \mu_1 + \gamma_1) + \mu_m(\mu_0 + \mu_1 + \gamma_1)[\mu_m\alpha\beta_1 M_{I_1} + \mu_0\alpha\beta_1 H_{I_1} + \alpha^2\beta_1^2 H_{I_1} M_{I_1}]}.
$$

Observe that $\mathcal{R}_1'^2 < \mathcal{R}_1^2$, hence, we have $\mathcal{R}_1'^2 = \mathcal{R}_1^2 - K_1$, for some $K_1 > 0$. Therefore, using Descartes' rule of sign, we see that all the roots of (11) are negative if $\mathcal{R}_1^2 - K_1 < 1$, or equivalently if $\mathcal{R}_1^2 < 1 + K_1$. Since the endemic equilibrium does not exist for $\mathcal{R}_1^2 < 1$, we conclude that the endemic equilibrium for the strain-1 only model is locally asymptotically stable when $1 < \mathcal{R}_1^2 < 1 + K_1$. The same procedure can be used to show that the endemic equilibrium for the strain-2 only model is unstable when $1 < \mathcal{R}_2^2 < 1 + K_2$, for some $K_2 > 0$.

Results and discussion

For our simulations, we use values of different parameters given in table 1.

Figures 2 and 3 show the combine effect of both populations when the dengue fever is in epidemic state. Figure 2 represents the dynamics of the mosquitoes population assuming that 94.5% of the population is susceptible, infected mosquitoes of type-1 and type-2 represent each 2.2% of the population and finally infected mosquitoes of type-12 represent 1.1% of the population. And similarly, Fig. 3 represents the dynamics of humans population assuming that 95% of the population is susceptible, infected humans of type-1 and type-2 represent each 4% of the population and finally infected humans

Table 1 Parameter descriptions and values with source

Parameter	Description	Value	Source
π_h	Recruitment rate of humans into susceptible class	3.914×10^{-3}	Varies
π_m	Recruitment rate of mosquitoes into susceptible class	45	Varies
β_1	Biting rate of mosquitoes with strain-1 dengue virus	0.4	[16]
β_2	Biting rate of mosquitoes with strain-2 dengue virus	0.5	Assumed
β_{12}	Biting rate of mosquitoes with strain-1 and strain-2 dengue viruses	0.3	Assumed
γ_1	Recovering rate of humans with strain-1 or strain-2 dengue infection	0.16667	[16]
γ_2	Recovering rate of humans that are superinfected	0.125	Assumed
α	Probability of transmission of dengue virus	0.75	Assumed
μ_0	Natural Mortality rate of humans	3.914×10^{-5}	[17]
μ_1	Mortality rate of humans due to strain-1 or strain-2 dengue virus	0.005	Assumed
μ_2	Mortality rate of humans due to super-infecion	0.01	Assumed
μ_m	Mortality rate of aedes aegypti mosquitoes	0.002	[18]

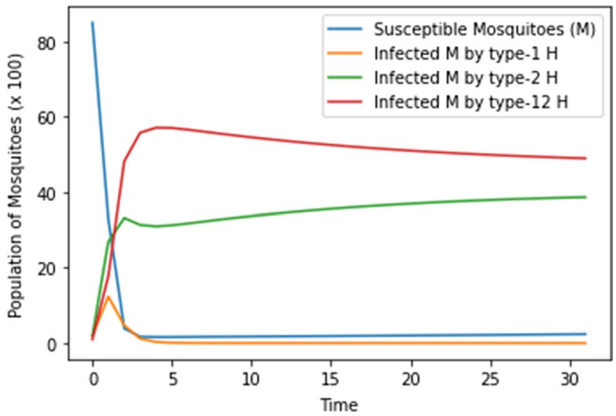

Fig. 2 Simulation of mosquito population

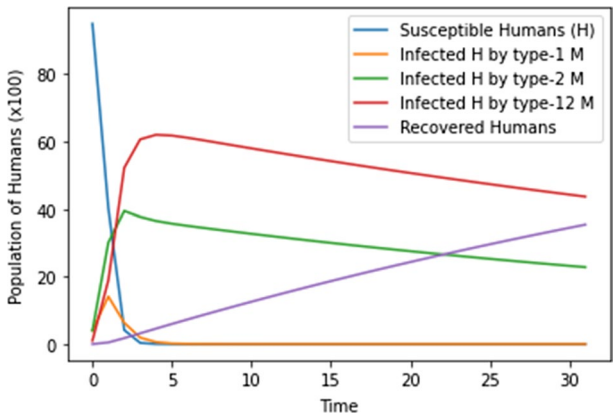

Fig. 3 Simulation of human population

of type-12 represent 1% of the population. The simulations show that with a high probability of transmission ($\alpha = 75\%$), both populations will get almost completely infected within the first week of the disease outbreak. It should also be noted that infected humans recover at a low rate.

In Figs. 4 and 5, the parameters are the same as in the previous simulations, but only the the probability of transmission is modified to $\alpha = 0.24$ (which is in accordance with literature). The populations' dynamics are the same but we observe that is takes a longer period for the majority of the population to get infected. So, by decreasing the value of α, we can slow down the spread of the disease. In this manner, the results are useful in forecasting disease trends and taking the appropriate control measures and strategies.

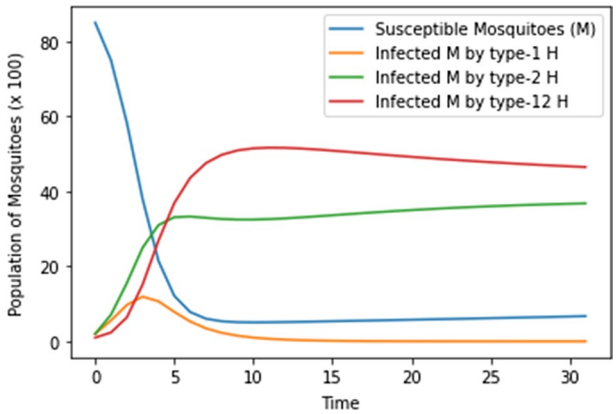

Fig. 4 Simulation of mosquito population with $\alpha = 24\%$

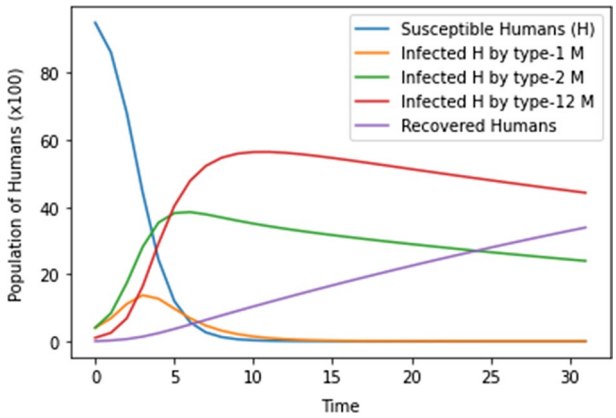

Fig. 5 Simulation of human population with $\alpha = 24\%$

Conclusion

In this paper, we formulated the dynamic transmission of two strains super-infection dengue. We obtained the basic reproduction number using next-generation matrix and carried out the stability analysis on the formulated problem. Based on qualitative analysis into the model, it was determined that Ξ_0 is locally asymptotically stable if $\mathcal{R}_1 < 1, \mathcal{R}_2 < 1$ and $\mathcal{R}_{12} < 1$. We obtained endemic equilibrium in strain-2 when $\mathcal{R}_2 > 1$. Our result revealed that decrease in the probability transmission rate will slows down the spread of the disease.

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Author Contributions
ASE drafted the problem and later was modified by MCA. ASE, MCA and SNN were involved on the write up. ASE and MCA carried out the mathematical analysis while SNN carried out the experimental numerical simulation. All the authors read and approved the final manuscript.

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Declarations

Competing interests
The authors declare that they have no competing interests

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References

1. Abidemi, A., Abd Aziz, M.I., Ahmad, R.: Vaccination and vector control effect on dengue virus transmission dynamics: modelling and simulation. Chaos Solitons Fractals **133**, 109648 (2020)
2. Massawe, L.N., Massawe, E.S., Makinde, O.D.: Temporal model for dengue disease with treatment. Adv. Infect. Dis. **5**, 21–36 (2015)
3. HamdanN, I., Adem Kilicman, A.: The development of a deterministic dengue epidemic model with the influence of temperature: a case study in Malaysia. Appl. Math. Model. **90**, 547–567 (2021)
4. Manore, C.A., Hickmann, K.S., Xu, S., Wearing, H.L., Hyman, J.M.: Comparing dengue and chikungunya emergence and endemic transmission *A. aegypti* and *A. albopictus*. J. Theor. Biol. **356**, 174–191 (2014)
5. Esteva, L., Vargas, C.: Analysis of a dengue disease transmission model. Math. Biosci. **150**, 131–151 (1998)
6. Adak, S., Jana, S.: A model to assess dengue using type 2 fuzzy inference system. Biomed. Signal Process. Control **63**, 102121 (2021)
7. Side, S., Noorani, S.M.: A SIR model for spread of dengue fever disease (Simulation for South Sulawesi, Indonesia and Selangor, Malaysia). World J. Model. Simul. **9**(2), 96–105 (2013)
8. Gubler, D.J., Kuno, G., Sather, G.E., Waterman, S.H.: A case of natural concurrent human infection with two dengue viruses. Am. J. Trop. Med. Hyg. **34**(l), 170–173 (1985)
9. Abidemi, A., Fatoyinbo, H.O., Asamoah, J.K.K., Muni, S.S: Evaluation of the efficacy of Wolbachia intervention on dengue burden in a population: a mathematical insight. In: 2022 International Conference on Decision Aid Sciences and Applications (DASA), Chiangrai, Thailand, pp. 1618–1627 (2022). https://doi.org/10.1109/DASA54658.2022.9765106
10. Abidemi, A., Fatoyinbo, H.O., Asamoah, J.K.K: Analysis of dengue fever transmission dynamics with multiple controls: a mathematical approach. In: 2020 International Conference on Decision Aid Sciences and Application (DASA), Sakheer, Bahrain, pp. 971–978 (2020). https://doi.org/10.1109/DASA51403.2020.9317064
11. Peijiang Liu, P., Anwarud, D., Zenab: Impact of information intervention on stochastic dengue epidemic model. Alex. Eng. J. **60**, 5725–5739 (2021)
12. Asamoah, J.K.K., Yankson, E., Okyere, E., Sund, G., Jina, Z., Jane, R.: Optimal control and cost-effectiveness analysis for dengue fever model with asymptomatic and partial immune individuals. Results Phys. (2021). https://doi.org/10.1016/j.rinp.2021.104919
13. Nipa, K.E., Sophia, R.J., Jang, Linda, J.S., Allen: The effect of demographic and environmental variability on disease outbreak for a dengue model with a seasonally varying vector population. Math. Biosci. **331**, 108516 (2021)
14. Agarwal, P., Singh, R., Rehman, A.: Numerical solution of hybrid mathematical model of dengue transmission with relapse and memory via Adam-Bashforth-Moulton predictor-corrector scheme. Chaos Solitons Fractals **143**, 110564 (2021)
15. Eegunjobi, A.S., Makinde, O.D.: Mathematical analysis of two strains of COVID-19 using SEIR model. J. Math. Fund. Sci. **54**(2), 211–232 (2022)
16. Syafruddin, S.S., Nooeami, S.M.: SEIR model for transmission of dengue fever in Malaysia. Int. J. Mod Phys Conf Ser **9**, 380–389 (2012)
17. Katri, P.: Modeling the transmission dynamics of the dengue virus. Open Access Dissertations. Paper 417 (2010)
18. Ferreira, C.P., Pinho, S.T.R., Esteva, L.: Modelling the dynamics of dengue real epidemics. Anais do CNMAC, 3 (2010)
19. Watmough, J., Drissche, P.V.: Reproduction numbers and sub-threshold endemic equilibria for compartment models of disease transmission. Math. Biosci. **180**, 29–48 (2002)

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