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Transmission Dynamic Model of Influenza Complicated By a Secondary Bacterial Infection In The Presence Of Control

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Abstract
Influenza virus infection represents a global threat causing seasonal outbreaks and pandemics, and as a result, it is associated with considerable morbidity and mortality worldwide despite the availability of vaccine and antiviral drugs. However, medical experts and published data has shown that deaths are largely caused by respiratory complications resulting from secondary bacterial infections, the most common being bacterial pneumonia. In order to understand the transmission and control dynamics of this infection in the presence of secondary bacterial infections, we formulated an eight compartmental mathematical model, which incorporated vaccination and treatment parameters into the deterministic model that studies the infection behaviour in the presence of secondary bacterial infections.
The mathematical analysis shows that the disease free and the endemic equilibrium point of the model exist. The model has disease free equilibrium point which is locally asymptotically stable and is globally asymptotically stable whenever the basic reproduction number is less than unity i.e. $(R_0 < 1)$ *and unstable when* $R_0 > 1$.
Numerical simulation was carried out by Maple 18 software using differential transformation method to investigate the effects of vaccine, transmission rates, asymptomatic progression rate and treatment rates on the dynamics of the disease.
Our results showed that increasing the rates of vaccination and influenza treatment rate has a significant effect of reducing influenza infection in both populations of the infected individuals and increased influenza treatment increases the temporarily-recovered population. The transmission rates (β_1 and β_2) have the effect of reducing the susceptible population while increasing influenza infection in both populations of the infected individuals. Increase in asymptomatic progression rate, ε, increases asymptomatic infection of influenza in the population, but lessen the number of people who are symptomatically infected with influenza. The bacteria-transmission rate β_3 also decreases the temporarily recovered population while increasing the bacteria-infected population. However, bacteria treatment decreases secondary infection in the population, and greatly increases the population of the fully recovered individuals.
The effect of transmission rates can be reversed when individuals take preventive measures to help slow the spread of germs that cause flu. Also, if screening programmes are organized for all individuals irrespective of whether they show symptoms or not, the infection status of all individuals would be known, and as such, the population of the asymptomatic infected individuals can be reduced, while their symptomatic infected population will be treated accordingly.

Keywords: *Influenza, Bacterial infection, Complication, Vaccination, Critical points, Basic reproduction number, Stability*

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I. Introduction

Influenza (also known as Flu) is a contagious respiratory illness caused by influenza viruses that infect the nose, throat and lungs. It can cause mild to severe illness and sometimes lead to death [7]. Influenza virus may be transmitted among humans in three ways: (1) direct contact with infected individuals; (2) contact with contaminated objects (called fomites) such as toys, doorknobs, etc.; (3) inhalation by virus-laden aerosols.

People who have influenza often feel some or all of these signs and symptoms: fever/chills, cough, sore throat, running/stuffy nose, muscle/body aches, headache, fatigue/tiredness and vomiting and diarrhoea (common in children). It should be noted that not everyone with flu have fever. The time from when a person is exposed to the virus to when symptoms begins is about 1 – 4 days, with an average of about 2 days [7]. However, approximately 33% of people with influenza are asymptomatic [4]. Most infected people recover within one to two weeks without requiring medical treatment. However, in the very young, the elderly, and those with other

serious medical conditions, infection can lead to severe complication of underlying condition (like asthma, diabetes, heart disease, etc.); secondary bacterial infections (like pneumonia, bronchitis, sinus, ear infection etc.); and death [22].

To prevent influenza, the influenza vaccine is recommended by the WHO and the United states CDC to the high-risk group such as children, the elderly, health care workers and people with chronic illness, or are immuno-compromised (such as people with HIV/AIDS) among others. It can also be prevented by everybody preventive actions (like staying away from people who are sick, covering coughs and sneezes and frequent hand washing) to help slow the spread of germs that cause respiratory (nose, throat and lungs) illness like flu [7]. There are influenza antiviral drugs (like Neuraminidase inhibitor, Oseltamivir, among others) that can be used to treat flu illness [7] [22].

Influenza virus infections are associated with considerable morbidity and mortality worldwide. In the US alone, despite the availability of vaccine and antiviral drugs, influenza causes approximately 200,000 serious infections that require hospitalization and 36,000 deaths each year. Influenza pandemics and epidemics which mostly occur annually in the fall or winter pose threats (such as missed work, cost of hospitalisation and medical treatment and increased deaths) to the human population [21]. As a result, it is important to understand to detail, the dynamics of this disease.

A number of works has been done on the spread of multiple strains of the influenza virus with immunity [1], [15], [17], [18]; on modelling the dynamics with different age groups [5], [9], [13], [19]. However, medical experts and published data has shown that deaths are largely caused by respiratory complications resulting from secondary bacterial infections, the most common being bacterial pneumonia [2], [3], [16]. In 2009, Handel *et al.* derived a mathematical model for a bacterial infection following influenza, which addressed the scientific problem of the infection thoroughly but with the parameters not known to details. In 2012, Chien *et al.* [8] proposed a model that looks into the contribution of antibacterial intervention on the co-infection of influenza and secondary bacterial infection. But their model is somewhat complex. Also Henneman *et al.* (2013) [12] proposed a model that studied bacterial infection following influenza. In the present study, we formulate a new model following the Oluyo and Adeyemi model [17], and the Henneman *et al.* model [12] to get a better insight into the dynamical transmission and control of influenza complicated by a secondary bacterial infection.

The rest of this work is organized as follows: Section 2 gives a full description of the model and shows a domain where the model is epidemiologically well posed. Section 3 provides the existence of equilibria including a derivation of the basic reproduction number and stability analysis of the equilibria. In Section 4, we perform numerical simulations of the model with graphical illustrations and their discussion, and give concluding remark in Section 5.

II. Model Formulation

The new model which sub-divides the total human population size at time t, denoted by $N(t)$, into susceptible humans $S(t)$, Vaccinated humans $V(t)$, Exposed humans $E(t)$, Asymptomatic infected humans $I_A(t)$, Symptomatic infected humans $I_S(t)$ Recovered from influenza and temporarily susceptible to bacterial infection $T(t)$, Humans infected with secondary bacterial infection $I_b(t)$ and Fully recovered humans $R(t)$. Hence, we have:

$$N(t) = S(t) + V(t) + E(t) + I_A(t) + I_s(t) + T(t) + I_b(t) + R(t).$$

The transmission and control of influenza Virus among human is governed by some basic epidemiological parameters. Susceptible individuals are recruited into the human population either by birth or immigration at a rate π, out of which a fraction ν is vaccinated and so, are protected against the infection, and the remaining fraction $(1-\nu)$ receives no vaccine. The vaccine wanes off at a rate ω and individuals of the vaccinated class return to susceptible class. When an infected individual, either asymptomatic or symptomatic, comes in contact with a susceptible human, the virus is passed onto the human and the person moves to the exposed class $E(t)$ at a rate β_1 and β_2 respectively (the model did not include the transmissions from virus laden aerosols).

The human natural and disease-induced death rates are denoted respectively as μ, δ_1, and δ_2. The average exposure period is $1/\rho$, after which a fraction ε of $\rho E(t)$ shows no symptom. Other parameters are as given in table 2.1.

The figure 2.1 below shows the dynamics of the model with the inflow and outflow on each compartment.

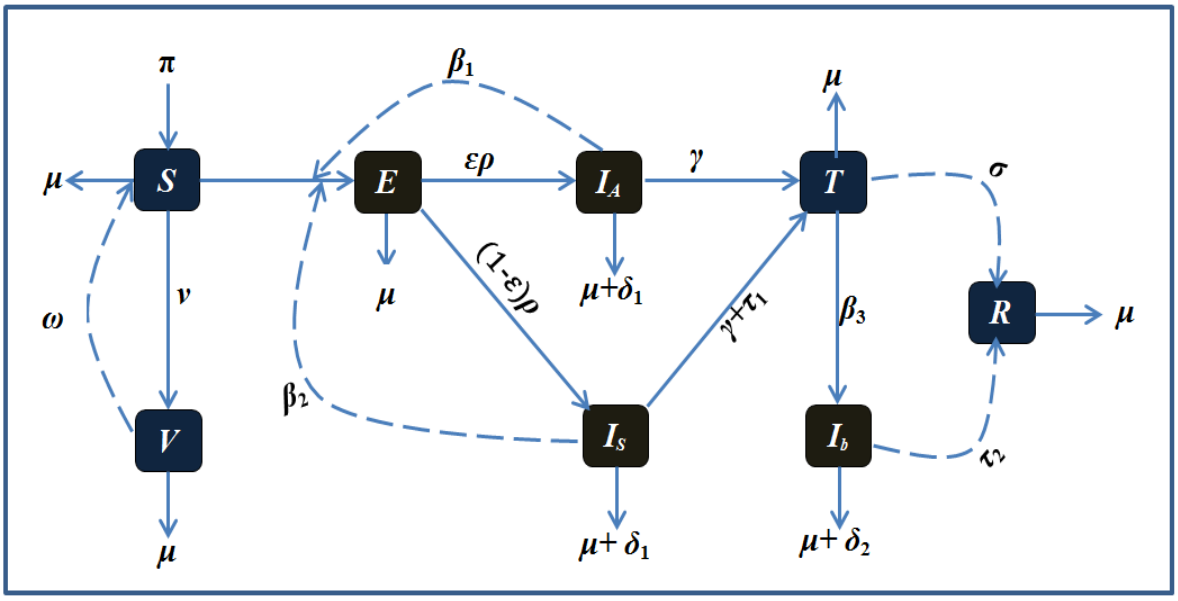

Figure 2.1: *The diagrammatic representation for the dynamics of influenza virus infection*

The model is formulated as a system of coupled ordinary differential equation as:

$$\begin{aligned}
\frac{dS}{dt} &= (1-v)\pi - \mu S - \left(\beta_1 I_A + \beta_2 I_S\right) S + \omega V \\
\frac{dV}{dt} &= v\pi - (\mu + \omega)V \\
\frac{dE}{dt} &= \left(\beta_1 I_A + \beta_2 I_S\right) S - (\mu + \rho)E \\
\frac{dI_A}{dt} &= \varepsilon\rho E - (\mu + \delta_1 + \gamma) I_A \\
\frac{dI_S}{dt} &= (1-\varepsilon)\rho E - (\mu + \delta + \gamma + \tau_1) I_S \\
\frac{dT}{dt} &= \gamma I_A + (\gamma + \tau_1) I_S - (\mu + \sigma + \beta_3 I_b) T \\
\frac{dI_b}{dt} &= \beta_3 I_b T - (\mu + \delta_2 + \tau_2) I_b \\
\frac{dR}{dt} &= \sigma T + \tau_2 I_b - \mu R;
\end{aligned} \tag{2.1}$$

together with the initial conditions:

$$S(t_0) = S_0,\ V(t_0) = V_0,\ E(t_0) = E_0,\ I_A(t_0) = I_{A0},\ I_S(t_0) = I_{S0},\ T(t_0) = T_0,\ I_b(t_0) = I_{b0},\ R(t_0) = R_0.$$

The state variables and parameters used for the transmission model are described in the following table:

State Variables and Parameters	Description
$S(t)$	Number of individuals susceptible to influenza infection at time t
$V(t)$	Number of individuals vaccinated against influenza infection at time t
$E(t)$	Number of individuals exposed to influenza infection at time t
$I_A(t)$	Number of asymptomatic infected individuals at time t
$I_S(t)$	Number of symptomatic infected individuals at time t
$T(t)$	Number of recovered individuals from influenza that are temporarily susceptible to bacterial infection at time t
$I_b(t)$	Number of individuals infected with secondary bacterial infection at time t
$R(t)$	Number of fully recovered individuals at time t
N	Total human population
π	Recruitment term of the susceptible individuals
v	Per capita rate of vaccination
ω	Per capita rate of vaccine wanes off

β_1	Rate of transmission from contact between susceptible and asymptomatic infected individuals
β_2	Rate of transmission from contact between susceptible and asymptomatic infected individuals
β_3	Rate of transmission of secondary bacterial infection
ρ	Per capita rates of progression from the exposed state to the infected states
ε	Fraction of the exposed individuals that are migrated to symptomatic infected
δ_1, δ_2	Disease-induced death rates for influenza and secondary bacterial infections respectively
μ	Natural death rate
γ	Natural recovery rate of the infected individuals from influenza
τ_1, τ_2	Per capita recovery rates due to treatment of influenza and bacterial infections respectively
σ	Progression rate from temporary susceptibility to full recovery state

Table 2.1: *Description of Variables and parameters used in the model*

Existence and Uniqueness of Solution

THEOREM 2.1 **[10]:** Let

$$\begin{aligned} x_1' &= f_1(x_1, x_2, \ldots x_n, t), x_1(t_0) = x_{10} \\ x_2' &= f_2(x_1, x_2, \ldots x_n, t), x_2(t_0) = x_{20} \\ &\vdots \\ x_n' &= f_n(x_1, x_2, \ldots x_n, t), x_n(t_0) = x_{n0}. \end{aligned} \tag{2.2}$$

Suppose D is the region in (n+1)-dimensional space (one dimension for t and n dimensions for the vector x). If the partial derivatives $\frac{\partial f_i}{\partial x_j}, i, j = 1,2,\ldots,n$ are continuous in

$$D = \{(x,t) : |t - t_0| \le a, |x - x_0| \le b\},$$

then there is a constant $\delta > 0$ such that there exists a unique continuous vector solution $\underline{x} = [x_1(t), x_2(t), \ldots, x_n(t)]$ in the interval $|t - t_0| \le \delta$.

THEOREM 2.2**:** Let

$$\begin{aligned} f_1 &= \frac{dS}{dt} = (1-\nu)\pi - \mu S - (\beta_1 I_A + \beta_2 I_S) S + \omega V; & S(t_0) &= S_0 \\ f_2 &= \frac{dV}{dt} = \nu\pi - (\mu + \omega)V; & V(t_0) &= V_0 \\ f_3 &= \frac{dE}{dt} = (\beta_1 I_A + \beta_2 I_S) S - (\mu + \rho)E; & E(t_0) &= E_0 \\ f_4 &= \frac{dI_A}{dt} = \varepsilon\rho E - (\mu + \delta_1 + \gamma) I_A; & I_A(t_0) &= I_{A_0} \\ f_5 &= \frac{dI_S}{dt} = (1-\varepsilon)\rho E - (\mu + \delta_1 + \gamma + \tau_1) I_S; & I_S(t_0) &= I_{S_0} \\ f_6 &= \frac{dT}{dt} = \gamma I_A + (\gamma + \tau_1) I_S - (\mu + \sigma + \beta_3 I_b) T; & T(t_0) &= T_0 \\ f_7 &= \frac{dI_b}{dt} = \beta_3 I_b T - (\mu + \delta_2 + \tau_2) I_b; & I_b(t_0) &= I_{b_0} \\ f_8 &= \frac{dR}{dt} = \sigma T + \tau_2 I_b - \mu R; & R(t_0) &= R_0, \end{aligned} \tag{2.3}$$

$$D = \{(S, V, E, I_A, I_S, T, I_b, R) : |S - S_0| \le a, |V - V_0| \le b, |E - E_0| \le c, |I_A - I_{A_0}| \le d, |I_S - I_{S_0}| \le e, |T - T_0| \le f, |I_b - I_{b_0}| \le g, |R - R_0| \le h, |t - t_0| \le i\}.$$

Then equation (2.3) has a unique solution.

***Proof*:**
We find the partial derivatives, evaluated at the origin thus:

$$\left.\frac{\partial f_1}{\partial S}\right|_{(0,0,0,0,0,0,0,0)} = -\mu \qquad \left.\frac{\partial f_2}{\partial S}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_3}{\partial S}\right|_{(0,0,0,0,0,0,0,0)} = 0$$

$$\left.\frac{\partial f_1}{\partial V}\right|_{(0,0,0,0,0,0,0,0)} = -\omega \qquad \left.\frac{\partial f_2}{\partial V}\right|_{(0,0,0,0,0,0,0,0)} = -(\mu+\omega) \qquad \left.\frac{\partial f_3}{\partial V}\right|_{(0,0,0,0,0,0,0,0)} = 0$$

$$\left.\frac{\partial f_1}{\partial E}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_2}{\partial E}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_3}{\partial E}\right|_{(0,0,0,0,0,0,0,0)} = -(\mu+\rho)$$

$$\left.\frac{\partial f_1}{\partial I_A}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_2}{\partial I_A}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_3}{\partial I_A}\right|_{(0,0,0,0,0,0,0,0)} = 0$$

$$\left.\frac{\partial f_1}{\partial I_S}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_2}{\partial I_S}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_3}{\partial I_S}\right|_{(0,0,0,0,0,0,0,0)} = 0$$

$$\left.\frac{\partial f_1}{\partial T}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_2}{\partial T}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_3}{\partial T}\right|_{(0,0,0,0,0,0,0,0)} = 0$$

$$\left.\frac{\partial f_1}{\partial I_b}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_2}{\partial I_b}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_3}{\partial I_b}\right|_{(0,0,0,0,0,0,0,0)} = 0$$

$$\left.\frac{\partial f_1}{\partial R}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_2}{\partial R}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_3}{\partial R}\right|_{(0,0,0,0,0,0,0,0)} = 0$$

$$\left.\frac{\partial f_4}{\partial S}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_5}{\partial S}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_6}{\partial S}\right|_{(0,0,0,0,0,0,0,0)} = 0$$

$$\left.\frac{\partial f_4}{\partial V}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_5}{\partial V}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_6}{\partial V}\right|_{(0,0,0,0,0,0,0,0)} = 0$$

$$\left.\frac{\partial f_4}{\partial E}\right|_{(0,0,0,0,0,0,0,0)} = \varepsilon\rho \qquad \left.\frac{\partial f_5}{\partial E}\right|_{(0,0,0,0,0,0,0,0)} = -(1-\varepsilon)\rho \qquad \left.\frac{\partial f_6}{\partial E}\right|_{(0,0,0,0,0,0,0,0)} = 0$$

$$\left.\frac{\partial f_4}{\partial I_A}\right|_{(0,0,0,0,0,0,0,0)} = (\mu+\delta+\gamma) \qquad \left.\frac{\partial f_5}{\partial I_A}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_6}{\partial I_A}\right|_{(0,0,0,0,0,0,0,0)} = \gamma$$

$$\left.\frac{\partial f_4}{\partial I_S}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_5}{\partial I_S}\right|_{(0,0,0,0,0,0,0,0)} = -(\mu+\delta_1+\gamma+\tau_1) \qquad \left.\frac{\partial f_6}{\partial I_S}\right|_{(0,0,0,0,0,0,0,0)} = \gamma+\tau_1$$

$$\left.\frac{\partial f_4}{\partial T}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_5}{\partial T}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_6}{\partial T}\right|_{(0,0,0,0,0,0,0,0)} = -(\mu+\sigma)$$

$$\left.\frac{\partial f_4}{\partial I_b}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_5}{\partial I_b}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_6}{\partial I_b}\right|_{(0,0,0,0,0,0,0,0)} = 0$$

$$\left.\frac{\partial f_4}{\partial R}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_5}{\partial R}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_6}{\partial R}\right|_{(0,0,0,0,0,0,0,0)} = 0$$

$$\left.\frac{\partial f_7}{\partial S}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_8}{\partial S}\right|_{(0,0,0,0,0,0,0,0)} = 0$$
$$\left.\frac{\partial f_7}{\partial V}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_8}{\partial V}\right|_{(0,0,0,0,0,0,0,0)} = 0$$
$$\left.\frac{\partial f_7}{\partial E}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_8}{\partial E}\right|_{(0,0,0,0,0,0,0,0)} = 0$$
$$\left.\frac{\partial f_7}{\partial I_A}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_8}{\partial I_A}\right|_{(0,0,0,0,0,0,0,0)} = 0$$
$$\left.\frac{\partial f_7}{\partial I_S}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_8}{\partial I_S}\right|_{(0,0,0,0,0,0,0,0)} = 0$$
$$\left.\frac{\partial f_7}{\partial T}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_8}{\partial T}\right|_{(0,0,0,0,0,0,0,0)} = \sigma$$
$$\left.\frac{\partial f_7}{\partial I_b}\right|_{(0,0,0,0,0,0,0,0)} = -(\mu+\delta_2+\tau_2) \qquad \left.\frac{\partial f_8}{\partial I_b}\right|_{(0,0,0,0,0,0,0,0)} = \tau_2$$
$$\left.\frac{\partial f_7}{\partial R}\right|_{(0,0,0,0,0,0,0,0)} = 0 \qquad \left.\frac{\partial f_8}{\partial R}\right|_{(0,0,0,0,0,0,0,0)} = -\mu$$

Obviously, $\left|\frac{\partial f_i}{\partial x_j}\right|, i, j = 1,2,...,8$ are continuous and bounded in

$$D = \left\{(S,V,E,I_A,I_S,T,I_b,R): |S-S_0| \le a, |V-V_0| \le b, |E-E_0| \le c, \left|I_A - I_{A_0}\right| \le d, \left|I_S - I_{S_0}\right| \le e, |T-T_0| \le f, \left|I_b - I_{b_0}\right| \le g, |R-R_0| \le h, |t-t_0| \le i\right\}.$$

Hence, following Derrick and Grossman [10] of theorem 2.1 above, the problem (2.3) has a unique solution and so the model (2.1) is both epidemiologically feasible and mathematically well posed.

III. Mathematical Analysis Of The Model

In this section we carry out qualitative analysis of the model (2.1) to investigate existence and stability of the steady states.

3.1. Existence of Equilibrium Points

Let $E = \left(S^*, V^*, E^*, I_A^*, I_S^*, T^*\right)$ represent any arbitrary equilibrium point of system (2.1) obtained by setting $\frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI_A}{dt} = \frac{dI_S}{dt} = \frac{dT}{dt} = 0$; i.e.

$$\begin{aligned}
(1-\nu)\pi - \mu S^* - \left(\beta_1 I_A^* + \beta_2 I_S^*\right) S^* + \omega V^* &= 0 \\
\nu\pi - (\mu+\omega)V^* &= 0 \\
\left(\beta_1 I_A^* + \beta_2 I_S^*\right) S^* - (\mu+\rho)E^* &= 0 \\
\varepsilon\rho E^* - (\mu+\delta_1+\gamma) I_A^* &= 0 \\
(1-\varepsilon)\rho E^* - (\mu+\delta_1+\gamma+\tau_1) I_S^* &= 0 \\
\gamma I_A^* + (\gamma+\tau_1) I_S^* - (\mu+\sigma+\beta_3 I_b^*) T^* &= 0 \\
\beta_3 I_b^* T^* - (\mu+\delta_2+\tau_2) I_b^* &= 0 \\
\sigma T^* + \tau_2 I_b^* - \mu R^* &= 0
\end{aligned} \qquad (2.4)$$

Disease-free Equilibrium Points, R_0

Disease-free equilibrium points are steady-state solutions in the absence of influenza virus infection (i.e. $I_A = I_S = I_b = 0$). Thus, the disease-free equilibrium point, E_0, for the influenza virus model (2.1) when $I_A = I_S = I_b = 0$ yields:

$$E_0 = \left(\frac{\pi}{\mu}\left[1-\nu+\frac{\omega\nu}{\mu+\omega} \right],\ \frac{\nu\pi}{\mu+\omega},\ 0,\ 0,\ 0,\ 0,\ 0,\ 0 \right) \qquad (2.5)$$

3.2. Derivation of Basic Reproduction Number, R_0

An important notion in epidemiological models is the basic reproduction number, usually denoted by R_0. It is a threshold value that is often used to measure the spread of a disease. It is defined as the number of secondary infections in humans that arise as a result of a single infected individual being introduced in a fully susceptible population. When $R_0 < 1$, it implies that on average an infectious individual infects less than one person throughout his/her infectious period and in this case the disease is wiped out. On the other hand, when $R_0 > 1$, then on average every infectious individual infects more than one individual during his/her infectious period and the disease persists in the population.

The derivation of basic reproduction number is essential in order to assess the local stability of the system (2.1). To do this, we employ the method of next generation matrix described by Driessche and Watmough [21].

We have the transmission and transition matrices to be given respectively as

$$\mathcal{F} = \begin{pmatrix} (\beta_1 I_A + \beta_2 I_S)S \\ 0 \\ 0 \\ \beta_3 IT \end{pmatrix} \text{ and } \qquad \mathcal{V} = \begin{pmatrix} (\mu+\rho)E \\ -\varepsilon\rho E + (\mu+\delta_1+\gamma)I_A \\ -(1-\varepsilon)\rho E + (\mu+\delta_1+\gamma+\tau_1)I_S \\ (\mu+\delta_2+\tau_2)I \end{pmatrix}.$$

The Jacobian matrices for $\mathcal{F}$ and $\mathcal{V}$ at DFE (E_0) are evaluated as follows:

$$F = D\mathcal{F}\big|_{E_0} = \begin{pmatrix} 0 & \frac{\pi\beta_1}{\mu}\left[1-\nu+\frac{\omega\nu}{\mu+\omega}\right] & \frac{\pi\beta_2}{\mu}\left[1-\nu+\frac{\omega\nu}{\mu+\omega}\right] & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \qquad \text{and}$$

$$V = D\mathcal{V}\big|_{E_0} = \begin{pmatrix} \mu+\rho & 0 & 0 & 0 \\ -\varepsilon\rho & \mu+\delta_1+\gamma & 0 & 0 \\ -(1-\varepsilon)\rho & 0 & \mu+\delta_1+\gamma+\tau_1 & 0 \\ 0 & 0 & 0 & \mu+\delta_2+\tau_2 \end{pmatrix}$$

$$F.V^{-1} = \begin{pmatrix} \frac{\pi\rho\left(1-\nu+\frac{\omega\nu}{\mu+\omega}\right)}{\mu(\mu+\rho)}\left[\frac{\varepsilon\beta_1}{\mu+\delta_1+\gamma}+\frac{(1-\varepsilon)\beta_2}{\mu+\delta_1+\gamma+\tau_1}\right] & \frac{\pi\beta_1}{\mu}\left[1-\nu+\frac{\omega\nu}{\mu+\omega}\right] & \frac{\pi\beta_2}{\mu}\left[1-\nu+\frac{\omega\nu}{\mu+\omega}\right] & 0 \\ \frac{}{(\mu+\delta_1+\gamma)} & \frac{}{(\mu+\delta_1+\gamma+\tau_1)} & & \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

Now, the basic reproduction number, which equals $\boldsymbol{\rho}(F.V^{-1})$, is obtained as the spectra radius (i.e. the dominant eigenvalue) of the product $F.V^{-1}$ thus:

$$R_0 = \frac{\pi\rho\left(1-\nu+\frac{\omega\nu}{\mu+\omega}\right)}{\mu(\mu+\rho)}.\left[\frac{\varepsilon\beta_1}{\mu+\delta_1+\gamma}+\frac{(1-\varepsilon)\beta_2}{\mu+\delta_1+\gamma+\tau_1}\right] \tag{2.7}$$

This quantity gives the basic reproduction number.

3.3. Local Stability of Disease-free Equilibrium Point

***THEOREM 3.1*:**

The disease-free equilibrium is locally asymptotically stable.

***Proof*:** The stability of the disease-free equilibrium is determined by the eigenvalues of the Jacobian matrix of the full system (2.1), evaluated at the disease-free equilibrium point, given by:

$$J\Big|_{E_0} = \begin{pmatrix} -\mu & \omega & 0 & -\frac{\pi\beta_1}{\mu}\left(1-\nu+\frac{\omega\nu}{\mu+\omega}\right) & -\frac{\pi\beta_2}{\mu}\left(1-\nu+\frac{\omega\nu}{\mu+\omega}\right) & 0 & 0 & 0 \\ 0 & -(\mu+\omega) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\mu+\rho) & \frac{\pi\beta_1}{\mu}\left(1-\nu+\frac{\omega\nu}{\mu+\omega}\right) & \frac{\pi\beta_2}{\mu}\left(1-\nu+\frac{\omega\nu}{\mu+\omega}\right) & 0 & 0 & 0 \\ 0 & 0 & \varepsilon\rho & -(\mu+\delta_1+\gamma) & 0 & 0 & 0 & 0 \\ 0 & 0 & (1-\varepsilon)\rho & 0 & -(\mu+\delta_1+\gamma+\tau_1) & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma & \gamma+\tau_1 & -(\mu+\sigma) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -(\mu+\delta_2+\tau_2) & 0 \\ 0 & 0 & 0 & 0 & 0 & \sigma & \tau_2 & -\mu \end{pmatrix}$$

.

The eigenvalues of this Jacobian matrix are obtained to be:

$$\lambda_1 = -\mu,\ \lambda_2 = -(\mu+\omega),\ \lambda_3 = -(\mu+\rho),\ \lambda_4 = -(\mu+\delta_1+\gamma),$$

$$\lambda_5 = -(\mu+\delta_1+\gamma+\tau_1),\ \lambda_6 = -(\mu+\sigma),\ \lambda_7 = -(\mu+\delta_1+\tau_2),\ \lambda_8 = -\mu;$$

Since all the roots are real distinct and negative, then the disease-free equilibrium point is locally asymptotically stable. This completes the proof.

3.4.1. Global Stability of Disease-free Equilibrium Point

Here, we explored the global asymptotic stability (GAS) property of the disease-free equilibrium point for the influenza model.

***THEOREM 3.2*:**

If $R_0 < 1$, then the disease-free equilibrium is globally asymptotically stable. Otherwise, it is unstable.

***Proof*:** This proof is based on the use of comparison theorem [14] using the comparison method. Thus we have:

$$\begin{pmatrix} \frac{dE}{dt} \\ \frac{dI_A}{dt} \\ \frac{dI_s}{dt} \\ \frac{dI_b}{dt} \end{pmatrix} = (F-V)\begin{pmatrix} E \\ I_A \\ I_s \\ I_b \end{pmatrix} - F_i\begin{pmatrix} E \\ I_A \\ I_s \\ I_b \end{pmatrix}$$

where $F - V$ is defined as

$$F-V=\begin{pmatrix} -(\mu+\rho) & \frac{\pi\beta_1}{\mu}\left(1-\nu+\frac{\omega\nu}{\mu+\omega}\right) & \frac{\pi\beta_2}{\mu}\left(1-\nu+\frac{\omega\nu}{\mu+\omega}\right) & 0 \\ \varepsilon\rho & -(\mu+\delta_1+\gamma) & 0 & 0 \\ (1-\varepsilon)\rho & 0 & -(\mu+\delta_1+\gamma+\tau_1) & 0 \\ 0 & 0 & 0 & -(\mu+\delta_2+\tau_2) \end{pmatrix}=\tilde{J}$$

$\tilde{J}$ can be rewritten in the form $\tilde{J} = M - D$, where

$$M=\begin{pmatrix} 0 & \frac{\pi\beta_1}{\mu}\left(1-\nu+\frac{\omega\nu}{\mu+\omega}\right) & \frac{\pi\beta_2}{\mu}\left(1-\nu+\frac{\omega\nu}{\mu+\omega}\right) & 0 \\ \varepsilon\rho & 0 & 0 & 0 \\ (1-\varepsilon)\rho & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix},$$

$$D=\begin{pmatrix} (\mu+\rho) & 0 & 0 & 0 \\ 0 & (\mu+\delta_1+\gamma) & 0 & 0 \\ 0 & 0 & (\mu+\delta_1+\gamma+\tau_1) & 0 \\ 0 & 0 & 0 & (\mu+\delta_2+\tau_2) \end{pmatrix}.$$

D is a diagonal matrix with positive diagonal elements and therefore it is a non-singular matrix, while M is the remainder. The eigenvalue of $\tilde{J}$ have negative real parts *iff* the spectra radius (i.e. the dominant eigenvalue) of the matrix $MD^{-1} < 1$ [7], where

$$MD^{-1}=\begin{pmatrix} 0 & \frac{\pi\beta_1}{\mu(\mu+\delta+\gamma)}\left(1-\nu+\frac{\omega\nu}{\mu+\omega}\right) & \frac{\pi\beta_2}{\mu(\mu+\delta+\gamma+\tau)}\left(1-\nu+\frac{\omega\nu}{\mu+\omega}\right) & 0 \\ \frac{\varepsilon\rho}{\mu+\rho} & 0 & 0 & 0 \\ \frac{(1-\varepsilon)\rho}{\mu+\rho} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

The eigenvalues of MD^{-1}, obtained by setting $|M D^{-1} - \lambda I| = 0$, where I is a 4 x 4 identity matrix, is given as the roots of the quartic equation:

$$\lambda^4-\frac{\pi\beta_1\left(1-\nu+\frac{\omega\nu}{\mu+\omega}\right)}{\mu(\mu+\delta_1+\gamma)}\frac{\varepsilon\rho}{\mu+\rho}\lambda^2-\frac{\pi\beta_2\left(1-\nu+\frac{\omega\nu}{\mu+\omega}\right)}{\mu(\mu+\delta_1+\gamma+\tau_1)}\frac{(1-\varepsilon)\rho}{\mu+\rho}\lambda^2=0$$

$$\Rightarrow \qquad \lambda^2\left\{\lambda^2-\frac{\pi\rho\left(1-\nu+\frac{\omega\nu}{\mu+\omega}\right)}{\mu(\mu+\rho)}\left[\frac{\varepsilon\beta_1}{\mu+\delta_1+\gamma}+\frac{(1-\varepsilon)\beta_2}{\mu+\delta_1+\gamma+\tau_1}\right]\right\}=0$$

$$\Rightarrow \qquad \lambda_{1,2}=0 \quad Or \quad \lambda_{3,4}=\pm\sqrt{\frac{\pi\rho\left(1-\nu+\frac{\omega\nu}{\mu+\omega}\right)}{\mu(\mu+\rho)}\left[\frac{\varepsilon\beta_1}{\mu+\delta_1+\gamma}+\frac{(1-\varepsilon)\beta_2}{\mu+\delta_1+\gamma+\tau_1}\right]}\ =\ \pm\sqrt{R_0}.$$

Obviously, the leading eigenvalue $\sqrt{R_0}$ is less than unity if $R_0 < 1$. In other words, the spectra radius of $MD^{-1} < 1$. Therefore, all the eigenvalue of $\tilde{J}$ have negative real parts.

Hence, the disease-free equilibrium point of the system (2.1) is globally asymptotically stable if $R_0 < 1$, and unstable if otherwise. This completes the proof.

3.4. Endemic Equilibrium Point, E_e

In addition to the disease-free equilibrium point E_0, we shall show that the model (2.1) has an endemic equilibrium point, E_e. The endemic equilibrium point is a positive steady state solution where the disease persists in the population (i.e. if $I_A \neq I_S \neq I_b \neq 0$). Therefore, solving the system (2.4) simultaneously gives the endemic equilibrium defined by:

$$E_e = \left(S^*,\ V^*,\ E^*,\ I_A^*,\ I_S^*,\ T^*,\ I_b^*,\ R^*\right); \tag{2.6}$$

where

$$S^* = \frac{1}{\mu}\left\{\pi\left(1-\nu+\frac{\omega\nu}{\mu+\omega}\right)-(\mu+\rho)C-\frac{\kappa(\mu+\delta_2+\tau_2)}{\mu+\sigma}I_b^*\right\};$$

$$V^* = \frac{\nu\pi}{\mu+\omega};$$

$$E^* = C;$$

$$I_A^* = \frac{\varepsilon\rho}{\mu+\delta_1+\gamma}C;$$

$$I_S^* = \frac{(1-\varepsilon)\rho}{\mu+\delta_1+\gamma+\tau_1}C;$$

$$T^* = \frac{1}{\mu+\sigma}\left\{\left(\frac{\varepsilon\gamma}{\mu+\delta_1+\gamma}+\frac{(1-\varepsilon)(\gamma+\tau_1)}{\mu+\delta_1+\gamma+\tau_1}\right)\rho C-(\mu+\delta_2+\tau_2)I_b^*\right\};$$

$$I_b^* = \frac{\mu+\sigma}{\mu+\delta_2+\tau_2}\left\{\frac{1}{\mu+\sigma}\left(\frac{\varepsilon\gamma}{\mu+\delta_1+\gamma}+\frac{(1-\varepsilon)(\gamma+\tau_1)}{\mu+\delta_1+\gamma+\tau_1}\right)\rho C-\frac{\mu+\delta_2+\tau_2}{\beta_3}\right\};$$

$$R^* = \frac{1}{\mu+\sigma}\left\{\frac{\sigma}{\beta_3}(\mu+\delta_2+\tau_2)+\frac{\tau_2(\mu+\sigma)}{\mu+\delta_2+\tau_2}\left[\frac{1}{\mu+\sigma}\left(\frac{\varepsilon\gamma}{\mu+\delta_1+\gamma}+\frac{(1-\varepsilon)(\gamma+\tau_1)}{\mu+\delta_1+\gamma+\tau_1}\right)\rho C-(\mu+\sigma)(\mu+\delta_2+\tau_2)\right]\right\},$$

with

$$C = \frac{1}{\mu+\rho}\left\{\pi\left(1-\nu+\frac{\omega\nu}{\mu+\omega}\right)-\frac{\mu(\mu+\rho)(\mu+\delta_1+\gamma)(\mu+\delta_1+\gamma+\tau_1)}{\varepsilon\rho\beta_1(\mu+\delta_1+\gamma+\tau_1)+(1-\varepsilon)\rho\beta_2(\mu+\delta_1+\gamma)}\right\}$$

Hence, an endemic equilibrium point E_e exists and is unique.

IV. Numerical Results And Discussion

The numerical simulation for the model was carried out by Maple 18 software using differential transformation method to show the effects of vaccination, recovery and treatment rates on the dynamics of influenza virus disease.

We used some of the parameter values compatible with the disease, obtained from literatures as given in the table 4.1 below, and by considering the initial conditions:

$S(0) = 500,\ V(0) = 175,\ E(0) = 250,\ I_A(0) = 100,\ I_S(0) = 150,\ T(0) = 200.$

Parameters	Description	Values	Sources

π	Recruitment term of the susceptible individuals	0.01547	Assumed
v	Per capita rate of vaccination	0.40	Estimated
ω	Per capita rate of vaccine wanes off	0.01	Estimated
β_1	Rate of transmission from contact between susceptible and asymptomatic infected individuals	0.30	Assumed
β_2	Rate of transmission from contact between susceptible and symptomatic infected individuals	0.25	[12]
β_3	Rate of transmission from contact between persons recovered from influenza and persons infected with bacterial infections	0.325	Assumed
ρ	Per capita rates of progression from the exposed state to the infected states	$^1/_{2.6} = 0.385$	[11]
ε	Fraction of the exposed individuals that are migrated to asymptomatic infected	0.33	Estimated
δ_1	Influenza-induced death rate	0.0005/day	[12]
δ_2	Death rate due to secondary bacterial infection	$^1/_{10} = 0.1$/day	[12]
μ	Natural death rate	0.009493	Assumed
γ	Natural recovery rate of the infected individuals	$½(^1/_7+^1/_{14})$	Estimated
τ_1	Per capita recovery rate due to treatment of influenza	$^1/_{2.4} = 0.417$	[11]
τ_2	Per capita recovery rate due to treatment of bacterial infection	$^1/_{18} = 0.0556$	[12]
σ	Progression rate from temporal recovery from influenza to full recovery state	$^1/_{14} = 0.0714$	[12]

Table 4.1: *Parameter values used for the model*

4.1. Presentation of Results

The results are given in Figures 4.1 – 4.12 to illustrate the system's behaviour for different values of the model's parameters.

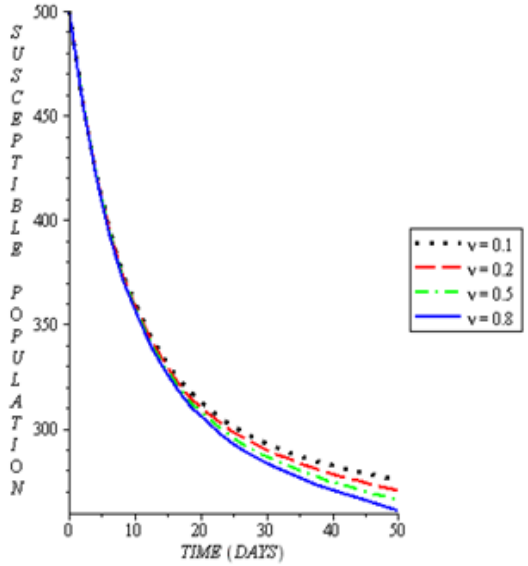

Fig. 4.1: *The behaviour of susceptible population for varied values of vaccination rate, v* (π = 0.01547, ω = 0.01, β_1 = 0.3, β_2 = 0.25, μ = 0.00493, δ =0 .1, $\gamma = {}^1/_2({}^1/_7+{}^1/_{14})$, τ = 1/2.4, ρ= 0.385, ε = 0.33).

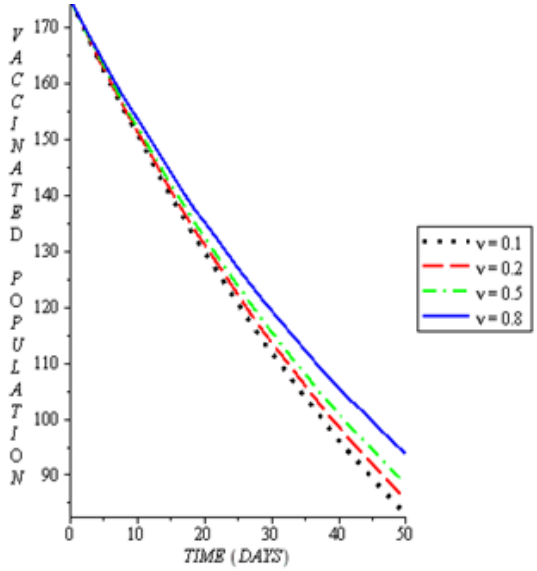

Fig. 4.2: *The behaviour of vaccinated population for varied values of vaccination rate, v* (π = 0.01547, ω = 0.01, β_1 = 0.3, β_2 = 0.25, μ = 0.00493, δ =0 .1, $\gamma = {}^1/_2({}^1/_7+{}^1/_{14})$, τ = 1/2.4, ρ= 0.385, ε = 0.33).

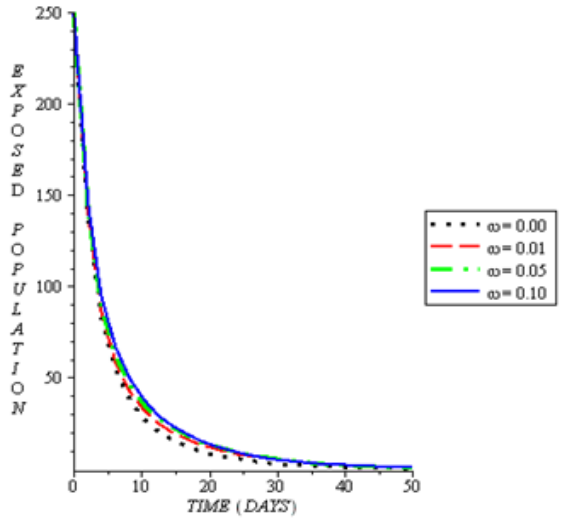

Fig. 4.3: *The behaviour of exposed population for varied values of vaccine wanes off rate, ω* (π = 0.01547, ν = 0.4, β_1 = 0.3, β_2 = 0.25, μ = 0.00493, δ =0 .1, $\gamma = {}^1/_2({}^1/_7+{}^1/_{14})$, τ = 1/2.4, ρ= 0.385, ε = 0.33).

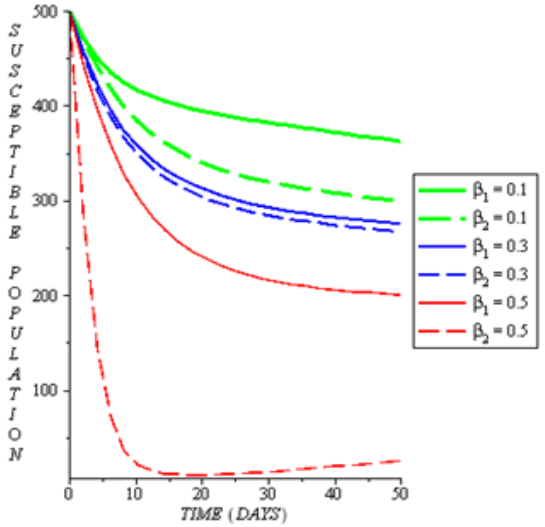

Fig. 4.4: *The behaviour of susceptible population for varied values of influenza transmission rates, β_1, β_2* (π = 0.01547, ν = 0.4, ω = 0.01, μ = 0.00493, δ =0 .1, $\gamma = {}^1/_2({}^1/_7+{}^1/_{14})$, τ = 1/2.4, ρ= 0.385, ε = 0.33).

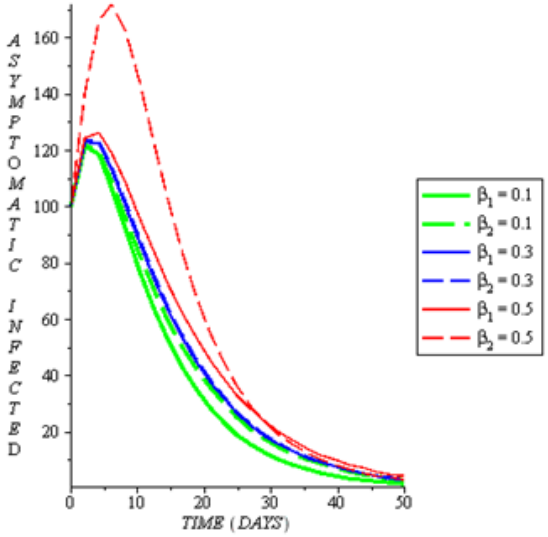

Fig. 4.5: *The behaviour of asymptomatic infected population for varied values of influenza transmission rates, β_1, β_2* (π = 0.01547, ν = 0.4, ω = 0.01, μ = 0.00493, δ =0 .1, $\gamma = {}^1/_2({}^1/_7+{}^1/_{14})$, τ = 1/2.4, ρ= 0.385, ε = 0.33).

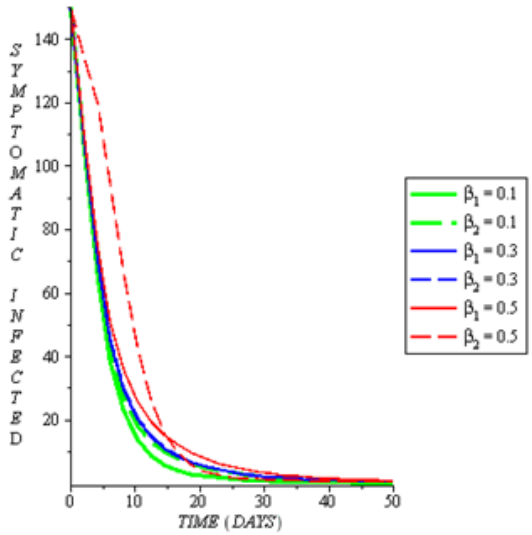

Fig. 4.6: *The behaviour of symptomatic infected population for varied values of influenza transmission rates, β_1, β_2* (π = 0.01547, ν = 0.4, ω = 0.01, μ = 0.00493, δ =0 .1, $\gamma = {}^1/_2({}^1/_7+{}^1/_{14})$, τ = 1/2.4, ρ= 0.385, ε = 0.33).

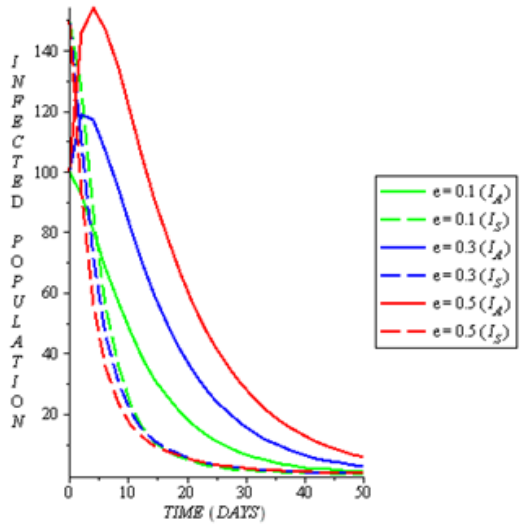

Fig. 4.7: *The behaviour of influenza infected populations for varied values of asymptomatic progression rate, ε (π = 0.01547, ν = 0.4, ω = 0.01, β_1 = 0.3, β_2 = 0.25, μ = 0.00493, δ =0 .1, $\gamma = {}^1/_2({}^1/_7+{}^1/_{14})$, τ = 1/2.4, ρ = 0.385).*

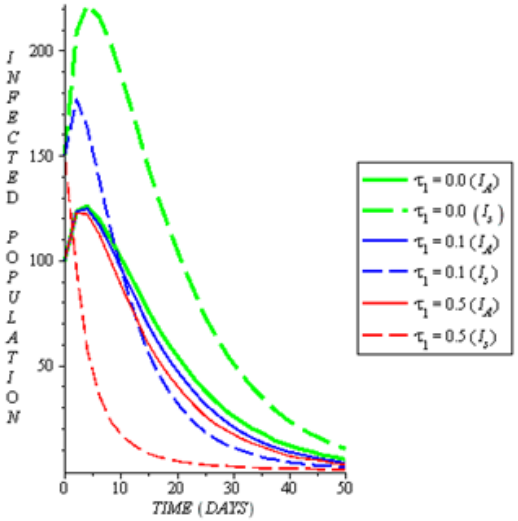

Fig. 4.8: *The behaviour of influenza infected populations for varied values of influenza treatment rate, τ_1 (π = 0.01547, ν = 0.4, ω = 0.01, β_1 = 0.3, β_2 = 0.25, μ = 0.00493, δ =0 .1, $\gamma = {}^1/_2({}^1/_7+{}^1/_{14})$, ρ = 0.385, ε = 0.33).*

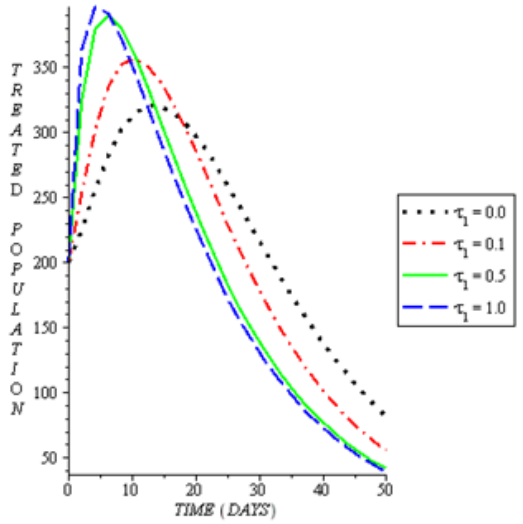

Fig. 4.9: *The behaviour of treated population for varied values of influenza treatment rate, τ_1 (π = 0.01547, ν = 0.4, ω = 0.01, β_1 = 0.3, β_2 = 0.25, μ = 0.00493, δ =0 .1, $\gamma = {}^1/_2({}^1/_7+{}^1/_{14})$, τ = 1/2.4, ρ = 0.385, ε = 0.33).*

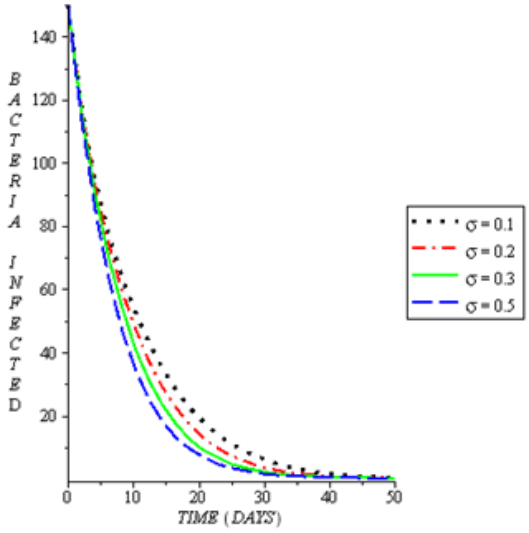

Fig. 4.10: *The behaviour of the bacteria infected population for varied values of progression rate to recovery, σ (π = 0.01547, ν = 0.4, ω = 0.01, β_1 = 0.3, β_2 = 0.25, μ = 0.00493, δ =0 .1, $\gamma = {}^1/_2({}^1/_7+{}^1/_{14})$, τ = 1/2.4, ρ = 0.385, ε = 0.33).*

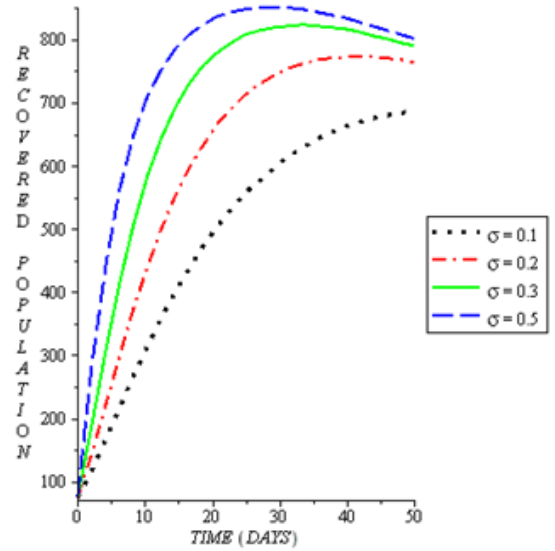

Fig. 4.11: *The behaviour of recovered population for varied values of progression rate to recovery, σ* ($\pi = 0.01547$, $\nu = 0.4$, $\omega = 0.01$, $\beta_1 = 0.3$, $\beta_2 = 0.25$, $\mu = 0.00493$, $\delta = 0.1$, $\gamma = {}^1/_2({}^1/_7 + {}^1/_{14})$, $\tau = 1/2.4$, $\rho = 0.385$, $\varepsilon = 0.33$).

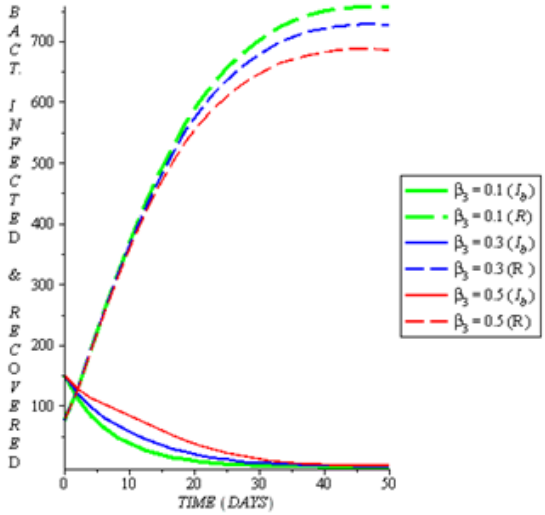

Fig. 4.12: *The behaviour of bacteria-infected and recovered populations for varied values of bacteria transmission rate, β_3* ($\pi = 0.01547$, $\nu = 0.4$, $\omega = 0.01$, $\beta_1 = 0.3$, $\beta_2 = 0.25$, $\mu = 0.00493$, $\delta = 0.1$, $\gamma = {}^1/_2({}^1/_7 + {}^1/_{14})$, $\rho = 0.385$, $\varepsilon = 0.33$)

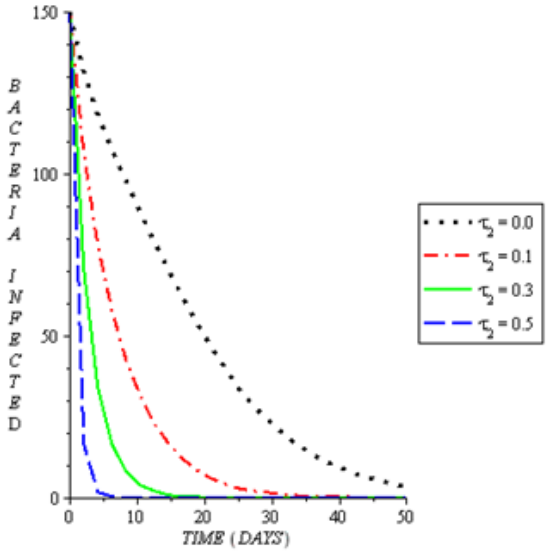

Fig. 4.13: *The behaviour of bacteria-infected population for varied values of secondary infection treatment rate, τ_2* ($\pi = 0.01547$, $\nu = 0.4$, $\omega = 0.01$, $\beta_1 = 0.3$, $\beta_2 = 0.25$, $\mu = 0.00493$, $\delta = 0.1$, $\gamma = {}^1/_2({}^1/_7 + {}^1/_{14})$, $\rho = 0.385$, $\varepsilon = 0.33$).

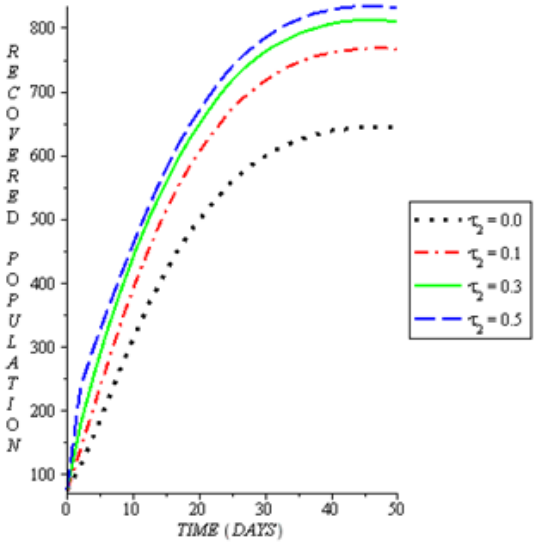

Fig. 4.14: *The behaviour of fully recovered population for varied values of secondary infection treatment rate, τ_2* ($\pi = 0.01547$, $\nu = 0.4$, $\omega = 0.01$, $\beta_1 = 0.3$, $\beta_2 = 0.25$, $\mu = 0.00493$, $\delta = 0.1$, $\gamma = {}^1/_2({}^1/_7 + {}^1/_{14})$, $\rho = 0.385$, $\varepsilon = 0.33$).

V. Discussion of Results

Figure 4.1 and figure 4.2 illustrate the effect of administering influenza vaccine at birth on the populations. From figure 4.1, we observed that increasing the vaccination rate, *v*, decreases the population of individuals that are susceptible to influenza, which in turn raises the population of individuals that are protected against the infection, i.e. the population of the vaccinated individuals increases as *v* increases. This can be seen as shown in figures 4.2. However from figure 4.3, which is the plot of exposed population against time for varied values of the rate of vaccine wane-off (ω), it was seen that the exposed population increases as ω

increases. This will in turn has the effect of increasing influenza infection in the population since more people are contracted and are exposed to the infection, and hence the need for more medical research in order to increase the efficacy and/or expiry duration of influenza vaccines. Also, while such research is on-going, influenza vaccines should be retaken for renewal sake and should not be once in a lifetime []. In these ways, the rate at which vaccines wane off will be greatly lowered, and the infection will be lowered in the population as well.

Figures 4.4 – 4.6 are plots that represent the effects of influenza transmission rates from asymptomatic and symptomatic infected individuals (β_1, β_2 respectively). From figures 4.4, which is the plots of susceptible population against time for varied values of β_1 and β_2, we observed a declining susceptible population as both rates β_1 and β_2 increases. From figures 4.5 and 4.6, which respectively represent the plot of asymptomatic and symptomatic infected populations against time for varied values of β_1 and β_2, it was seen that increase in β_1 and β_2 results into a corresponding increase in the population of asymptomatic and symptomatic infected individuals.

From figure 4.7, we have the plot of influenza infected population against time when the asymptomatic progression rate, ε, is varied. We observed that the more the people develop asymptomatic case of the infection the more the population of the asymptomatic infected individuals. On the other hand, the more the people develop asymptomatic case of influenza the lesser the people are symptomatically infected of the infection.

Also, we investigated the effect of influenza treatment rate (τ_1) on the populations. These effects are shown by figures 4.8 and 4.9. From figure 4.8, the plot of infected (asymptomatic and symptomatic) populations against time when τ_1 is varied, we observed that for the asymptomatic case, infection decreases just very slightly as τ_1 increases, while for the symptomatic case, there is a great deal of decrease in infection as τ_1 increases. This difference in the two cases can be attributed to the fact that only the symptomatic infected individuals are treated and not the asymptomatic infected individuals. Figure 4.9 is a plot of treated (recovered-from-influenza, temporarily-susceptible-to-bacterial-infection) population against time for varied values of τ_1. It was seen that the treated population increases initially as τ_1 increases, but with time, due to the fact that those treated are temporarily prone/susceptible to secondary bacterial infection within a period of 28days of recovery from influenza (after which some individuals progress to full recovery state, while some that contract bacteria within this time are secondarily infected with bacterial infection), the treated population at the long run decreases with time as τ_1 increases.

From figure 4.10, the plot of bacteria-infected population against time for varied values of progression rate to full recovery state (σ), it was noticed that the bacteria-infected population decreases with time as the rate at which treated individuals gain full recovery increases. In other words, the more the treated individuals develop immunity to fully recover, the lesser the number of individuals that would be secondarily infected with bacterial infection. Figure 4.11 is a graph of the recovered population against time for varied values of σ, and it shows that the recovered population increases as σ increases.

Furthermore, the effects of secondary infection parameters are investigated and the results displayed in figures 4.12 to 4.14. From figure 4.12, which represent the graph of bacteria-infected and recovered populations (combined) against time when the values of bacteria transmission rate, β_3, varies, it was shown that bacterial infection increases as β_3 increases, whereas, the recovered population decreases as β_3 increases. This is a complication posed by any secondary bacterial infection to influenza infection and must be controlled in order to minimize the number of deaths due to the said complication. Figure 4.13 and 4.14 are plots that show the effect of the treatment of bacterial infection (τ_2) on the populations. Figure 4.13 shows that there is a decline in secondary infection if the treatment of such infection is administered and is increased. Consequently, the population of the fully recovered individuals increases as depicted by figure 4.14, i.e. increasing the treatment rate of secondary bacterial infection results to an increase in the population of the fully recovered individuals.

VI. Conclusion

In this paper, we have formulated and analysed a compartmental model for influenza virus infection complicated by secondary bacterial infection. The total human population was divided into eight compartments: susceptible, vaccinated, exposed, asymptomatic infected, symptomatic infected, temporarily recovered, bacteria-infected and fully recovered sub-populations. We established a region where the model is epidemiologically feasible and mathematically well-posed. The existence and stability of a disease-free equilibrium point as well as the endemic equilibrium point were determined.

The numerical simulations were performed to see the effects of vaccine, transmission rates, asymptomatic progression rate and treatment rates on the dynamics of the disease. Our results showed that increasing the rates of vaccination and influenza treatment rate has a significant effect of reducing influenza infection in both populations of the infected individuals and increased influenza treatment increases the temporarily-recovered population (although, initially). The transmission rates (β_1 and β_2) have the effect of reducing the susceptible population while increasing influenza infection in both populations of the infected

individuals. Increase in asymptomatic progression rate, ε, increases asymptomatic infection of influenza in the population, but lessen the number of people who are symptomatically infected with influenza. The bacteria-transmission rate β_3 also decreases the temporarily recovered population while increasing the bacteria-infected population. However, bacteria treatment decreases secondary infection in the population, and greatly increases the population of the fully recovered individuals.

The effect of transmission rates can be reversed when individuals take preventive measures (like staying away from people who are sick, covering coughs and sneezes, and frequent hand washing) to help slow the spread of germs that cause respiratory illness (particularly flu). Also, in order to keep the progression rate to full recovery (σ) on the increase, patients who newly recovered from influenza should be isolated for a period of 28 days so as to prevent them from contracting any secondary infection due to bacteria. Furthermore, if screening programmes are organized for all individuals irrespective of whether they show symptoms or not, the infection status of all individuals would be known, and as such, the population of the asymptomatic infected individuals can be reduced, while their symptomatic infected population will be treated accordingly. Influenza antiviral drugs such as Neuraminidase inhibitor, Oseltamivir, etc. can be used to treat flu illness.

These control measures will greatly reduce the transmission of the influenza virus infection. However, efforts should be intensified in developing improved vaccines with higher efficacy and longer expiry duration for influenza virus disease as this would facilitate the stimulation of the immune system in producing antibodies against influenza virus infection.

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