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MATHEMATICAL MODELLING OF HIV/AIDSCONTROL USING VACCINATION STRATEGY

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Abstract: *HIV/AIDS is now a global pandemic and has eating deep into every states nation and continents. The need to control this world devastating disease in human history becomes very necessary and crucial. Vaccination has curbs some human disease in the past and will do same for HIV/AIDS. We develop a deterministic mathematical models using MATLAB 2007 for our simulation sand found the basic Reproduction number* R_0 *. The local and global stability of the diseases free and endemic equilibrium were conducted.*

Keywords: *HIV/AIDS, Basic Reproduction, Vaccine, Vaccination waning rate, imperfect.*

1 INTRODUCTION

An HIV vaccine is a vaccine which would either protect individuals who do not have HIV from contracting that virus, or may have a therapeutic effect for person who have or later contract HIV/AIDS. Currently, there is no effective HIV vaccine but many research project managing clinical trials seek to create one. This creates a lot ineffective vaccine in the society, but on a long run effective vaccine may be possible.

Human body can defend itself against HIV that is why certain individual can remain a symptomatic for decades after HIV infection. One HIV vaccine RV-144 was tried in Thailand at the beginning of 2003 and there was a positive result in 2009. Many trails have shown no efficacy, including the STEP Study and HVTN 505 trial, Healy, (2013).

Alternative medical treatments to a vaccine do exist. Highly Active Antiretroviral Therapy (HAART) has been highly beneficial to many HIV-infected individuals since its introduction in 1996. When the protease inhibitor based HAART initially became available. HAART allows the stabilization of the patient's symptoms and viremia, but they do not cure the patient of HIV, nor of the symptoms of AIDS. HAART does nothing in preventing the spread of HIV by people with undiagnosed infections. Therefore an HIV vaccine is generally considered as the most likely and perhaps the only way by which AIDS pandemic can be halted. However, after over 20 years of research, HIV -1 remains a difficult target for a vaccine. There are a number of factors that cause development of an HIV vaccine to differ from the development of other classical vaccines in existences.

(i) Classic vaccines mimic natural immunity against re-infection generally seen in individuals recovered from infection; there are almost no recovered AIDS patients.

(ii) Most vaccines protect against disease not against infection; HIV infection may remain latent for long period before causing AIDS.

(iii) Most effective vaccines are whole killed or live attenuated organism killed HIV-1 does not

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retain antigen and the use of a live retrovirus vaccine raise safety issues.

(iv) Most vaccine protect against infections through mucosal surfaces of the respiratory or gastrointestinal tract: the great majority of HIV infection is through a genital tract.

Blower et al (2001) worked on live attenuated HIV vaccines, predicting the trade off between efficacy and safety. In this study they stated that live attenuated HIV vaccine (LAHVs) could be extremely effective in preventing individual against infection with wild strains, but may not be completely save as this live vaccines could cause AIDS in some vaccinated individuals. Anderson et al (2007) presented a mathematical model to simulate the impact of various partially effective preventive HIV vaccination scenarios and change in risk behaviour within the population after receiving the vaccinesAnderson and Hanson (2005) worked on potential public health impact of imperfect HIV type I, vaccines was considered and analyzed. McLean and Blower (1993) worked on imperfect vaccine and herd immunity to HIV. Analytic and modelling techniques were used to investigate the effect of different levels of efficacy and coverage of a prophylactic vaccine on herd immunity and the possibility of eradicating the epidemic. Anderson and Garneth (1996) worked on low efficacy HIV vaccine potential for community based intervention programs. Garnett (1998) studied the influence of behavioural heterogeneity on the population level, impact of potential prophylactic HIV-1 vaccines: Bogard and Kuntz (2002) considered the impact of a partially effective HIV vaccine on a population of intravenous drug users in Bangkok, Thailand. Stover et al (2002) studied the epidemiological impact of an HIV/AIDs vaccine in developing country. The study used two different models applied to three different epidemic settings to examine the impact of vaccines with various characteristic on HIV incidence.

2 MATHEMATICAL MODEL FORMULATION.

The development of our model is based on the following assumptions that,

1. The diseases HIV/AIDS is killing continuously
2. Individual who contact this disease will definitely die of the disease if untreated or on control drug.
3. There is no vaccine with 100% efficacy to prevent HIV/AIDS
4. The available vaccines are imperfect; and so the vaccine will wane with time.

The model development is based on the assumptions that;

i. The HIV/AIDS disease is killing its victims continuously.
ii. There is no immediate curative agent for the disease
iii. That individual who contacted the disease will eventually die of the disease.
iv. That there is no vaccine with 100% efficacy rate to prevent HIV/AIDS
v. The available vaccine will wane with time.

Based on the ideas from assumption we develop a set of ordinary differential equation model about the dynamic of HIV/AIDS and some biological intervention.

We describe the interaction as; the susceptible class (S) recruited individual with rate (π) into the susceptible class and μ representing the natural death rate with the susceptible population as a result of public health campaign rate δ_1, if the vaccine is perfect, then the vaccinated individual is also the susceptible class. The waning rate of the vaccine at the rate θ, will lead the vaccinated individual into the exposed population. The exposed class has the natural death rate as μ and in contact with infected individual will be contacted at the rate ϕ an infected individual without treatment

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move to AIDS class (A) and the individual will definitely with time dies of HIV/AIDS. All the compartment reduces with natural death rate μ. The AIDS class increases the rate (η) of infected people.

Table I. State variable

$S(t)$	Number of susceptible at time t
$V(t)$	Vaccinated individual at time t
$E(t)$	Exposed individuals at time $t.$
$I(t)$	Infected individuals at time t
$A(t)$	Full blown AIDS class

Table II. Parameter descriptions.

π	Population recruited into the susceptible class.
μ	Per capita death rate (Nature death)
δ_1	Preventive Vaccination rate in the population
θ	Waning rate of the vaccine
θ_1	Vaccination efficacy rate
η	Rate of progression to full blown AIDS
u	Contact rate
ϕ	Progression rate of latent individual to infectious class.

VACCINATED INDIVIDUALS WITHOUT MUCH PUBLIC HEALTH CAMPAINGNS AND THERAPY

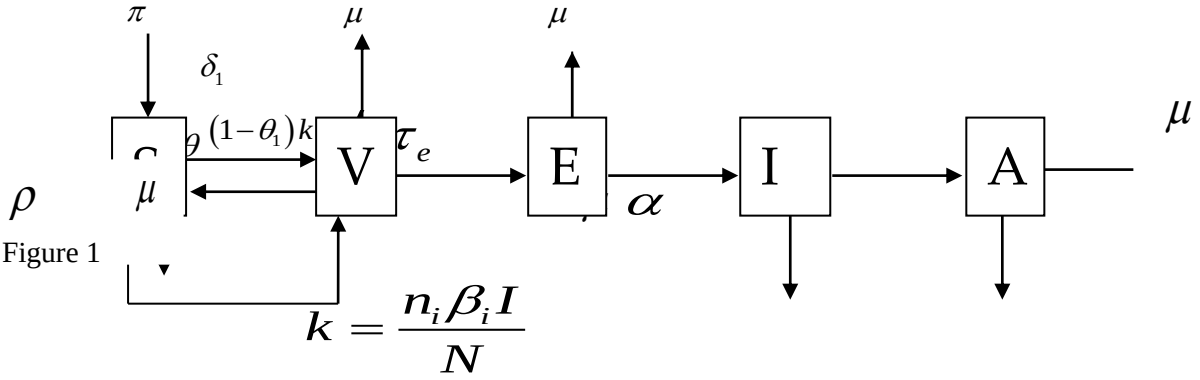

Figure 1

$$N = S + V + E + I + A$$

The models are;

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$$\frac{dS}{dt} = \pi + \delta_1 V - \theta S - \rho S - \mu S$$
$$\frac{dV}{dt} = \rho S + \theta S - \delta_1 V - (1-\theta_1) kV - \mu V$$
$$\frac{dE}{dt} = (1-\theta_1) kV - \phi E - \mu E \qquad (1)$$
$$\frac{dI}{dt} = \phi E - \tau_C I - \mu I$$
$$\frac{dA}{dt} = \tau_C I - (\alpha + \mu) A$$
$$S(0) > 0,\ V(0) > 0\ E(0) > 0\ I(0) > 0\ A(0) > 0$$
$$N = S + V + E + I + A$$

This gives

$$\frac{dN}{dt} = \pi + \delta_1 V - \theta S - \rho S - \mu S$$
$$+\rho s + \theta S - \delta_1 V - (1-\theta_1) Vk - \mu V$$
$$+(1-\theta_1) kV - \phi E - \mu E$$
$$+\phi E - \tau_e I - \mu I$$
$$+\tau_e I - \alpha A - \mu A$$
$$\frac{dN}{dt} = \pi - \alpha A - \mu S - \mu E - \mu I - \mu A$$
$$\frac{dN}{dt} = \pi - \alpha A - \mu N = 0$$
$$\Rightarrow \pi - \mu N = 0$$
$$\therefore \quad N = \frac{\pi}{\mu} \qquad (2)$$

At a point where there is no disease at all, the total population became

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$$N(t) \to \frac{\pi}{\mu} \text{ at } t \to \infty$$

$$\Rightarrow N(0) \geq \frac{\pi}{\mu} \qquad (3)$$

This then gives the following feasible invariant region

$$\Delta_1 = (S,V,E,I,A) \in R^5 \qquad (4)$$

3. MODEL ANALYSIS

We study the qualitative nature of the model (1) to understand the effect of vaccination only without public health campaign and therapeutic dozes on the eradication of the disease (HIV/AIDS), from the population. We start by determine the invariant region and show that all the solutions to (1) are positive at any $t > 0$

4. INVARIANT REGION

Theorem: The solutions of the model (1) are feasible at any $t > 0$ if they enter the invariant region Δ_1.

Proof: let $\Delta_1 = (S,V,E,I,A) \in R^5_+$ be only solution of the model system (1) with the non-negative initial conditions from (3) we have that in the absence of the disease

$$N \leq \pi - \mu N$$

$$N + \mu N \leq \pi \qquad (5)$$

Solve by finding the integrating factor

$$(IF) = e^{\int \mu dt} = e^{\mu t}$$

Multiply the integrating factor with all the terms in (5), we have

$$Ne^{\mu t} + \mu N e^{\mu t} \leq \pi e^{\mu t}$$

$$\frac{d}{dt}\left(Ne^{\mu t}\right) \leq \pi e^{\mu t}$$

Integrating

$$Ne^{\mu t} \leq \frac{\pi}{\mu} e^{\mu t} + C$$

Where C is the constant of integration

$$N \leq \frac{\pi}{\mu} + ce^{-\mu t}$$

At $t = 0 \qquad N(0) = N_0$

$$N_o - \frac{\pi}{\mu} \leq C$$

Therefore

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$$N \le \frac{\pi}{\mu} + \left(N_0 - \frac{\pi}{\mu}\right)e^{-\mu t} \qquad (6)$$

$$0 \le N \le \frac{\pi}{\mu} \text{ as } t \longrightarrow \infty$$

Birkhof and Rota (1982)

Therefore $\frac{\pi}{\mu}$ is the carry capacity. We let $Kp_2 = \frac{\pi}{\mu}$ where Kp_2 is the carrying capacity of the system, as the total population approach Kp_2, the feasible solution of the model enter the region,

$$\left\{\Delta_1 = (S, V, E, I, A) \in R^5_+\right\}$$

$$S > 0, V > 0, E > 0, I > 0 \text{ and } A > 0, N \le \frac{\pi}{\mu}$$

In this Δ_1 the model is biologically feasible and epidemiologically well posed. If $N \le \frac{\pi}{\mu}$ then every solution with the initial condition in Δ_1 remains in the region at all time $t > 0$. If $N > \frac{\pi}{\mu}$ then $N < 0$, then the population will reduce to the carrying capacity. Kp_2

5. POSITIVITY OF SOLUTION

Since the models are well posed and epidemiologically feasible in the region Δ_1. Using the same method as described in the general model, we show that the models are positive at all time $t > 0$.

Theorem:

Let the initial values of the models be $\left\{S(0) \ge 0,\ V(0) \ge 0,\ E(0) \ge 0,\ I(0) \ge 0 \ \text{and}\ A(0) \ge 0\right\} \in R^5_+$, then the solution set $S(t), V(t), E(t), I(t)$ and $A(t)$ of the system (1) is positive at all time $t \ge 0$

Proof:

From the first equation of the model (1) we have

$$\frac{dS}{dt} = \pi + \delta_1 V - \theta S - \rho S - \mu S$$

We have that

$$\frac{dS}{dt} = \pi + \delta_1 V - \theta S - \rho S - \mu S \ge -(\theta + \rho + \mu)S$$

Therefore we have that,

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$$\frac{\dot{S}}{S} \geq -(\theta+\rho+\mu)$$

$$\frac{dS}{S} \geq -\left((\theta+\rho)+\mu\right)dt$$

$$\ln S \geq -\left((\theta+\rho)+\mu\right)t + c$$

Where c is the constant of integration, apply the initial condition we have

$$S(t) \geq S(0)e^{-((\theta+p)+\mu)t} \geq 0 \qquad (7)$$

Since $(\theta+\rho+\mu)>0$

Therefore the solution is positive at all time. $t \geq 0$

From the second equation of (1), we have

$$\frac{dV}{dt} = \rho S + \theta S - \delta_1 V - (1-\theta_1)Vk - \mu V$$

We discovered that

$$\frac{dV}{dt} = \rho S + \theta S - \delta_1 V - (1-\theta_1)kV - \mu V \geq -\left(\delta_1 + (1-\theta_1)\right)kV - \mu V$$

$$\frac{dV}{dt} \geq -\left(\delta_1 + (1-\theta_1)k\right)V - \mu V$$

$$dV \geq -\left(\left(\delta_1 + (1-\theta_1)k\right)+\mu\right)Vdt$$

$$\frac{dV}{V} \geq -\left(\left(\delta_1 + (1-\theta_1)k\right)+\mu\right)dt$$

Integrating, we have

$$\ln V \geq -\left(\delta_1 + (1-\theta_1)+\mu\right)t + C_2$$

$$V(t) \geq V(0)e^{-(\delta_1+(1-\theta_1)+\mu)t} \geq 0$$

Since

$$\delta_1 + (1-\theta_1) \geq 0 \qquad (8)$$

And $\mu \geq 0$

Considering the third equation of (1) we have

$$\frac{dE}{dt} = (1-\theta_1)kV - \phi E - \mu E \geq -(\phi+\mu)E$$

$$\frac{dE}{E} \geq -(\phi+\mu)dt$$

$$\ln E \geq -(\phi+\mu)t + C$$

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$$E(t) \geq E(0)e^{-(\phi+\mu)t} \geq 0 \qquad (9)$$

Since $(\phi+\mu)$ is greater than zero and $\mu \geq 0$

Consider the fourth equation in the system.

$$\frac{dI}{dt} = \phi E - \tau_C I - \mu I$$

We let

$$\frac{dI}{dt} = \phi E - \tau_C I - \mu I \geq -(\tau_C + \mu) I$$

$$\frac{dI}{dt} \geq -(\tau_C + \mu) I$$

$$\frac{dI}{I} \geq -(\tau_C + \mu) dt$$

$$\ln I \geq -(\tau_C + \mu)t + C_3$$

$$I(t) \geq I(0)e^{-(\tau_C+\mu)t} \geq 0 \qquad (10)$$

Since $\tau_C + \mu \geq 0$ therefore $I(t) \geq 0$

From the fifth equations of the model we have

$$\frac{dA}{dt} = \tau_C I - (\alpha+\mu)A \geq -(\alpha+\mu)A$$

Therefore

$$\frac{dA}{dt} \geq -(\alpha+\mu)A$$

$$\Rightarrow \quad \frac{dA}{A} \geq -(\alpha+\mu)dt$$

$$In\, A \geq -(\alpha+\mu)t + C_4$$

Where C_4 is the constant of integration, therefore applying the initial condition, we have

$$A(t) \geq A(0)e^{-(\alpha+\mu)t} \geq 0 \qquad (11)$$

Since $(\alpha+\mu) \geq 0$

This shows that all the solutions of (1) are all positive at all time $t > 0$.

We have shown that the model is positive and invariant in the region $\Delta_1 \in R^5_{+,}$ we now determine the existence of the disease free equilibrium point to enable us calculate the Basic reproduction number. R

6. EXISTENCE OF DISEASE FREE EQUILIBRIUM POINT

From the region $\Delta_1 = (S, V, E, I, A)$ in the absence of the disease, we have that $V, E, I, A = 0$, we then have

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$$\pi - \mu S = 0$$

$$S^* = \frac{\pi}{\mu} \qquad (12)$$

Hence $E^* = \left(S^*, V^*, E^*, I^*, A^*\right) = \left(\frac{\pi}{\mu}, 0, 0, 0, 0\right)$

7. BASIC REPRODUCTION NUMBER

We employ the method used in Watmount (2009); the next generation method,we therefore generate the matrix F and V^{-1} by rewriting the model with the infected classes, (E, I, A) and follows by the rest.

The system then, becomes.

$$\frac{dE}{dt} = (1-\theta_1)Vk - \phi E - \mu E$$
$$\frac{dI}{dt} = \phi E - \tau_e I - \mu I$$
$$\frac{dA}{dt} = \tau_e I - \alpha A - \mu A \qquad (13)$$
$$\frac{dS}{dt} = \pi - \delta_1 V - \theta S - \rho S - \mu S$$
$$\frac{dV}{dt} = \rho S + \theta S - \delta_1 V - (1-\theta_1)kV - \mu V$$

$$\mathrm{k} = \frac{n_1 \beta_1 I}{N}$$

$$F_i = \begin{pmatrix} \frac{(1-\theta) n_i \beta_i I}{N} \\ 0 \\ 0 \end{pmatrix} \text{ Where } i = 1,2,3 \qquad (14)$$

$$V_i = \begin{pmatrix} \phi E + \mu E \\ -\phi E + \tau_e I + \mu I \\ -\tau_e I + \alpha A + \mu A \end{pmatrix} \qquad (15)$$

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$$F=\begin{pmatrix}(1-\theta)\frac{n_1\beta_1}{N} & \frac{n_1\beta_2}{N} & \frac{n_3\beta_3}{N}\\ 0 & 0 & 0\\ 0 & 0 & 0\end{pmatrix}$$

$$V=\begin{pmatrix}(\phi+\mu) & 0 & 0\\ -\phi & \tau_c+\mu & 0\\ 0 & -\tau_c & \alpha+\mu\end{pmatrix}$$

$$V^{-1}\begin{pmatrix}\frac{1}{\phi+\mu} & 0 & 0\\ \frac{\phi}{(\phi+\mu)(\tau_c+\mu)} & \frac{1}{\tau_c+\mu} & 0\\ \frac{\phi\tau_c}{(\phi+\mu)(\tau_c+\mu)(\alpha+\mu)} & \frac{\tau_c}{(\tau_c+\mu)(\alpha+\mu)} & \left(\frac{1}{\alpha+\mu}\right)\end{pmatrix}$$

By computing FV^{-1} we have

$$\begin{pmatrix}(1-\theta)\frac{n_1\beta_1}{N} & \frac{n_2\beta_2}{N} & \frac{n_3\beta_3}{N}\\ 0 & 0 & 0\\ 0 & 0 & 0\end{pmatrix}\begin{pmatrix}\frac{1}{\phi+\mu} & 0 & 0\\ \frac{\phi}{(\phi+\mu)(\tau_c+\mu)} & \frac{1}{\tau_c+\mu} & 0\\ \frac{\phi\tau_c}{(\phi+\mu)(\tau_c+\mu)(\alpha+\mu)} & \frac{\tau_c}{(\tau_c+\mu)(\alpha+\mu)} & \frac{1}{\alpha+\mu}\end{pmatrix} \quad (16)$$

This gives;

$$M_0=\begin{pmatrix}\frac{(1-\phi)n_1\beta_1}{\phi+\mu}+\frac{n_2\beta_2\phi}{(\phi+\mu)(\varepsilon+\mu)}+\frac{n_3\beta_3\phi\tau_C}{(\phi+\mu)(\tau_C+\mu)(\alpha+\mu)}, & \frac{n_2\beta_2}{\tau_C+\mu}+\frac{n_3\beta_3\tau_C}{(\tau_C+\mu)(\alpha+\mu)}, & \frac{n_3\beta_3}{(\alpha+\mu)}\\ 0 & 0 & 0\\ 0 & 0 & 0\end{pmatrix}$$

We therefore find the Eigen values of $M_{0,}$ we get,

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$$\lambda_1 = \frac{(1-\phi)n_1\beta_1}{\phi+\mu} + \frac{n_2\beta_2\phi}{(\phi+\mu)(\varepsilon+\mu)} + \frac{n_3\beta_3\phi\tau}{(\phi+\mu)(\tau_1+\mu)(\alpha+\mu)} \qquad \lambda_2 = 0, \quad \lambda_3 = 0$$

Therefore the dominant Eigen values is now our basic reproduction number giving as

$$R_{02} = \frac{(1-\theta_1)n_1\beta_1}{(\phi+\mu)} + \frac{\phi n_2\beta_2}{(\phi+\mu)(\varepsilon+\mu)} + \frac{\phi\eta_c n_3\beta_3}{(\phi+\mu)(\tau_1+\mu)(\alpha+\mu)}$$

Therefore

$$R_{02} = \frac{(1-\theta_1)(\varepsilon+\mu)(\tau_1+\mu)(\alpha+\mu)n_1\beta_1 + \phi(\tau_1+\mu)(\alpha+\mu)n_2\beta_2 + (\phi+\mu)\phi\tau_c n_3\beta_3}{(\phi+\mu)(\varepsilon+\mu)(\tau_1+\mu)(\alpha+\mu)} \quad \text{... (17)}$$

This R_{02} is the largest eigenvalues for the system (1) with vaccination as the only control strategy for the control of HIV/AIDS. The threshold quantity R_{02} is the effective reproduction number of (1) for HIV/AIDS infection in a population with vaccination strategy. It measures the average number of new infection generated by a single infected individual introduced into the completely susceptible population, in the presence of vaccination. (Anderson et-al 1995).

8. LOCAL STABILITY OF DISEASE FREE EQUILIBRIUM (DFE)

Local stability of DFE point is studied or investigated using our model system (1) given by $E_0 = (S,0,0,0,0) = \left(\frac{\pi}{\mu},0,0,0,0\right)$, at the disease free equilibrium point.

Theorem:

The vaccination induced DFE of the model is locally asymptotically stable if $R_{02} < 1$ and unstable if $R_{02} > 1$.

Proof: We shall use two approaches in trying to show our point, through the behaviour of our basic reproduction number and through the variation matrix method.

Epidemiologically speaking, theorem implies that HIV/AIDS can be eliminated from the population when $R_{02} < 1$ if the model are at the basic of attraction of disease free equilibrium E_0. This means that if $R_{02} < 1$, on average an infected individual produce less than one new infected individual into the population over the course of its infection period and the infection will definitely die out of the population, that means, the infection cannot grow. From equation (17) for our R_{02} to be less than one, this will be possible when θ (which implies vaccination) and C which implies public health (campaign) are increases without bound and as a result decrease the effect of β_1, β_2, β_3 and decrease the rate of exposure of infected individual during sex by using effective condom. Use of effective campaign and taking necessary medicine against HIV/AIDS, since the class are already infected.

Conversely if $R_{02} > 1$ then each infected individual produce on the average more than one new infection and the disease invade the population. This situation can be realized easily when we try to assess the contribution of *E*, *I*, *A*, in terms of β_1, β_2 and β_3 from (17) respectively.

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This means that

$$R_{021} = \frac{(1-\theta_1)n_1\beta_1}{\phi+\mu} \qquad (18)$$

$$R_{022} = \frac{\phi n_2\beta_2}{(\phi+\mu)(\varepsilon+\mu)} \qquad (19)$$

$$R_{023} = \frac{\phi\tau_c n_3\beta_3}{(\phi+\mu)(\tau_1+\mu)(\alpha+\mu)} \qquad (20)$$

Where $R_{020} = R_{021} + R_{022} + R_{023}$

From (18), (19), and (20), it is very obvious that $R_{021} > R_{022} > R_{023}$.

this implies that *E* contribute more on the transmission dynamic of HIV/AIDS since they are not aware of the condition and if they did not use condom effectively as it was the behaviour of many that undergo a very risky behaviour, followed by the infected class I, which keep the disease endemic in the population through $n_1\beta_1$ and $n_2\beta_2$ compared to $n_3\beta_3$ which are very much aware of their status.

For this model system to approach a steady state in the absence of infection the susceptible class will approach $\frac{\pi}{\mu}$ and β_2 and β_3 will be equal to zero respectively.

The condition can only be achieved if each infective is ready to take on only one sexual partner at a time and the rate at which those in the exposed classed are very ready to take possible precaution.

Alternatively, the variation matrix M_0 of the model system corresponding to E_0 (*DFE*) can also be used to study the local stability of the disease free equilibrium.

Consider the model system and let

$$\frac{dS}{dt} = \delta_1 V + \left(\lambda - (\theta+\rho+\mu)\right)S = H_1(S,V,E,I,A)$$

$$\frac{dV}{dt} = (\rho+\theta)S - \left(\delta_1 + (1-\phi_1)k + \mu\right)V = H_2(S,V,E,I,A)$$

$$\frac{dE}{dt} = (1-\theta_1)kV - (\phi+\mu)E = H_3(S,V,E,I,A) \qquad (20)$$

$$\frac{dI}{dt} = \phi E - (\tau_c+\mu)I = H_4(S,V,E,I,A)$$

$$\frac{dA}{dt} = \tau_c I - (\alpha+\mu)A = H_5(S,V,E,I,A)$$

Where we let

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$$t_1 = \pi - (\rho + \mu)$$
$$t_2 = \rho + \theta$$
$$t_3 = \delta_1 + (1 - \theta_1) K + \mu \qquad ..(21)$$
$$t_4 = 1 - \theta_1$$
$$t_5 = \phi + \mu$$
$$t_6 = \tau_c + \mu$$
$$t_7 = \alpha + \mu$$

The new model become

$$H_1 = \delta_1 V + -t_1 S + \pi = H_1(S, V, E, I, A)$$
$$H_2 = t_2 S - t_3 V = H_2(S, V, E, I, A)$$
$$H_3 = t_4 kV - t_5 E = (S, V, E, I, A) \qquad (22)$$
$$H_4 = \phi E - t_6 I = (S, V, E, I, A)$$
$$H_5 = \tau_c I - t_7 A = H_5(S, V, E, I, A)$$

$$M_0 = \begin{pmatrix} -t_1 & \delta_1 & 0 & 0 & 0 \\ t_2 & -t_3 & 0 & 0 & 0 \\ 0 & t_4 k & -t_5 & 0 & 0 \\ 0 & 0 & \phi & -t_6 & 0 \\ 0 & 0 & 0 & \tau_c & -t_7 \end{pmatrix} \qquad ..(23)$$

The characteristic equation corresponding to M_0 is shown in the appendix, since it is too large to be enclosed within.

$$\lambda^5 - a_1\lambda^4 - a_2\lambda^3 - a_3\lambda^2 - a_4\lambda + a_5 = 0$$

From the characteristic equation corresponding to M_0 we have

$$f(\nu) = (\nu_1 - (\theta + p + \mu))(\nu_2 + \delta + (1 - \theta)k - \mu)$$
$$(\nu_3 + \theta + \mu)(\nu_4 + \tau_c + \mu)(\nu_5 + \alpha + \mu) = 0$$

Where ν are the Eigen values

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$$\begin{aligned} v_1 &= -(\rho+\mu) \\ v_2 &= -(\delta+(1-\theta)k-\mu) \\ v_3 &= -(\mu) \\ v_4 &= -(\eta+\mu) \\ v_5 &= -(\alpha+\mu) \end{aligned} \qquad (24)$$

Therefore since all the Eigen values of the characteristics equation have negative real parts, then we can conclude that, the disease free equilibrium is locally asymptotically stable.

9. THE ENDEMIC EQUILIBRIUM POINT OF THE MODELS

From (1) and R_{02} given as

$$R_{02} = \frac{(1-\phi)n_1\beta_1}{\phi+\mu} + \frac{\phi n_2\beta_2}{(\phi+\mu)(\varepsilon+\mu)} + \frac{\phi\eta_c n_3\beta_3}{(\phi+\mu)(\tau_c+\mu)(\alpha+\mu)}$$

We equate the right hand side of (4.2.1) to zero, we can obtain the unique endemic equilibrium of the (4.2.1) as $E^* = (S^*, V^*, E^*, I^*, A^*)$ which exists for $R_{02} > 1, S^*, V^*, E^*, A^*$ and satisfies the following relations.

$$s^* = \frac{\pi(\delta_1+(1-\theta)k_i+\mu)}{(\theta+\rho+\mu)(\delta_1+(1-\theta_1)k_i+\mu)+\delta_1(\rho+\theta)} \qquad (25)$$

$$v^* = \frac{(\rho+\theta)\pi(\delta_1+(1-\theta_1)k_i+\mu)}{(\delta_1+(1-\theta_c)k_i+\mu)((\theta+\rho+\mu)(\delta_1+(1-\theta)k_i+\mu)+\delta_1(\rho+\theta)} \qquad (26)$$

$$e^* = \frac{\pi(p+\theta)\delta_1+(1-\theta_1)k_i+\mu)((\alpha+\mu)(1-\theta_1)+\eta_c)i}{(\delta_1+(1-\theta_1)k_i+\mu)((\theta+\rho+\mu)(\delta_1+(1-\theta_1)k_i+\mu)+\delta_1(\rho+\theta))} \qquad (27)$$

$$(\alpha+\mu)(\theta+\mu)$$

$$a^* = \frac{\eta_c i}{\alpha+\mu}$$

And

$$R_{02} = \frac{(1-\theta)n_1\beta_1}{\phi+\mu} + \frac{\phi n_2\beta_2}{(\phi+\mu)(\varepsilon+\mu)} + \frac{\phi\eta_c x_3\beta_3}{(\phi+\mu)(\tau_c+\mu)(\alpha+\mu)}. \qquad (28)$$

Where i is given as;

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$$i = \frac{\pi\phi(\rho+\theta)(\delta_1+\mu)(\alpha+\mu)\eta_c}{(\delta_1+\mu)\left((\theta+\rho+\mu)(\delta_1+\mu)+\delta_1(\rho+\theta)\right)(\alpha+\mu)(\theta+\mu)} \quad (29)$$

10 NUMERICAL SIMULATION

Here we illustrate the numerical simulation of the model (1) using the set of parameter values as given in the main model.

We use the Mat lab 7.1 programming language. The parameter values is as given in the following table

Table 1:

Parameters	Parameter values	Sources
π	50,000	Oluwaseun (2008)
δ_1	0.013	Oluwaseun (2008)
θ	0,025	Oluwaseun (2008)
μ	0.0001	Hem (2015)
θ_1	0.50	Liu(2008)
ϕ	0.7230	Christiana (2015)
τ	0.987	Christiana (2015)
c	5.00	Christiana (2015)
α	0.45	Hem (2015)

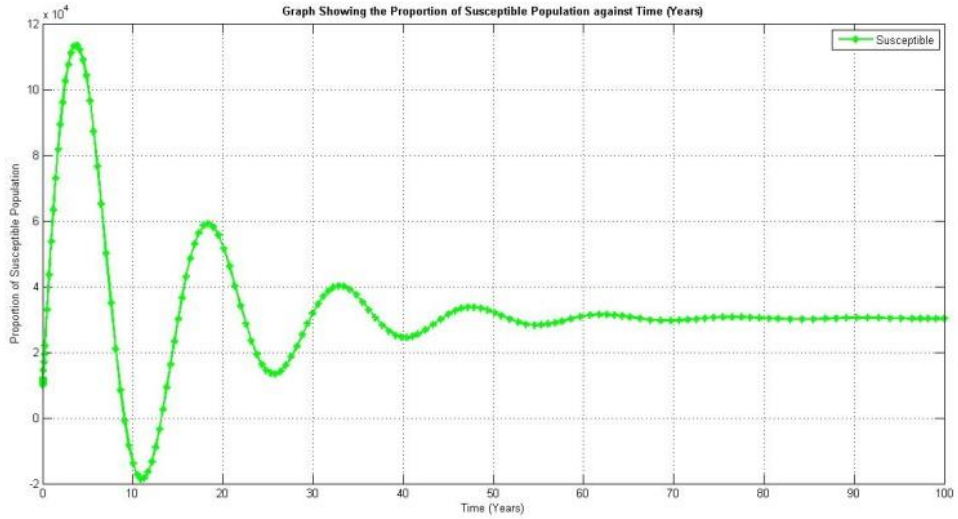

FIG 5.2.1: PROPORTION OF SUSCEPTIBLE WITH TIME.

Figure 5.2.1 shows the variation of the susceptible population with time using the varying parameters values $\pi = 50,000,\ \delta_1 = 0.013,\ \rho = 0.025,\ \mu = 0.0001\ \theta_1 = 0.50,\ \phi = 0.7230,\ c = 5.00,\ \alpha = 0.45\ \tau = 0.987$, using the public health campaign and vaccination as the control strategies. It is seen from the graph that initially the susceptible population rise and attained an equilibrium position and as a result of effective campaign and the effect of the vaccine those that will be susceptible to the infection decreases but not reaching zero rises again but this time due

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to waning rate of the vaccine after some time, the susceptible population become stable as R_{02} is greater than unity. The susceptible will now be transferred to the vaccinated class and some will leave the population through natural death rate (μ).

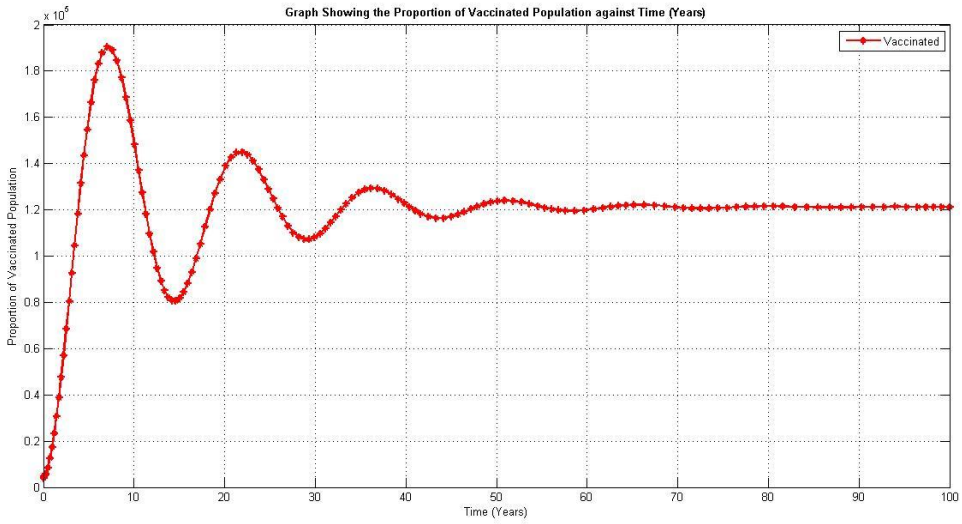

FIGURE 5.2.2: PROPORTION OF VACCINATED WITH TIME.

Figure 5.2.2 shows the variation of the vaccinated individual with time. It is seen that the individual recruited from susceptible class are vaccinated indicating the rise in the graph and attaining an equilibrium position, the sharp decreases is as a result of the waning rate of the vaccine after some period of about 15 years remaining stable, the waning effect of the vaccine move some individuals into the exposed class indicating that the infection is still maintained within the population.

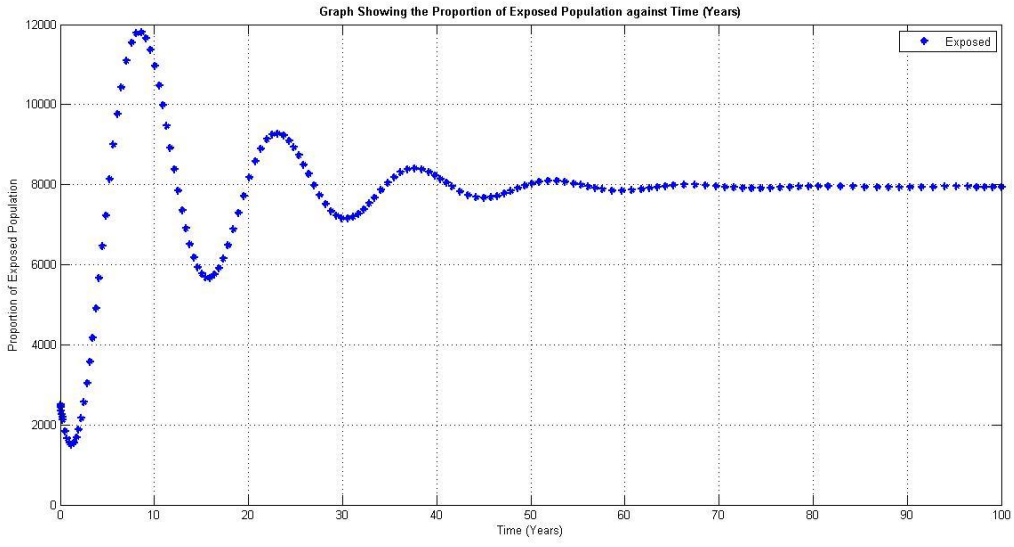

FIGURE 5.2.3: PROPORTION OF EXPOSED WITH TIME.

Figure 5.2.3 shows the proportion of the exposed individual with time. Initially the population of the exposed individual decreased as a result of the effect of the vaccine coverage level, but vaccine not being able to protect

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individual from the virus witness a sudden increase into the population of those who will be exposed to the infection this is due to the waning rate of the vaccine. Individual presented themselves to be vaccinated because of public health campaign sensitizing them on the benefit of vaccination with the parameters: $\pi = 50,000,\ \delta_1 = 0.013,\ \rho = 0.025,\ \mu = 0.0001\ \theta_1 = 0.50,\ \phi = 0.7230,\ \tau = 0.987,\ c = 5.00,\ \alpha = 0.45$.

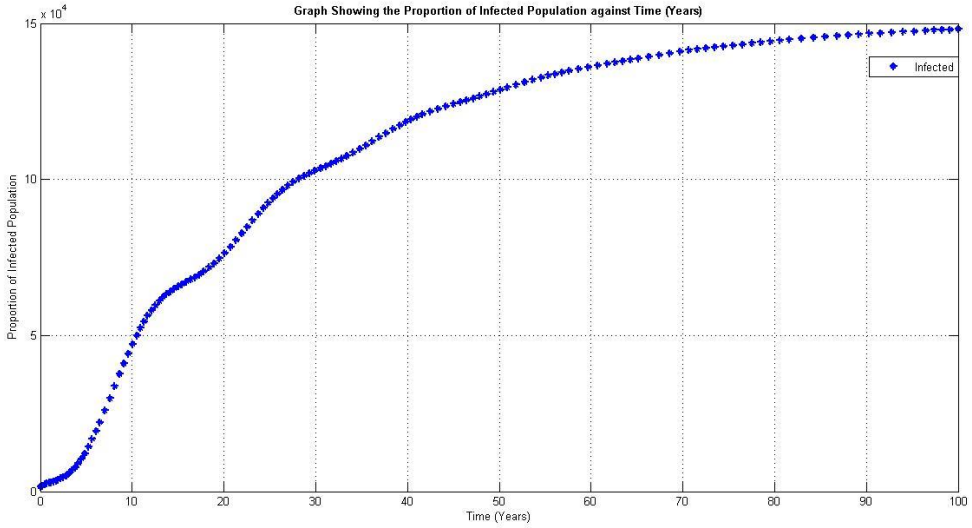

FIGURE 5.2.4: PROPORTION OF INFECTED WITH TIME.

Figure 5.2.4 shows the proportion of the infected class with time. It shows that individuals as a result of waning effect of the vaccine and the individual is not on any therapy, increase without bound. The sluggish increase in the population is a result of public health campaign urging them to be discipline in their life style but this alone has no effect on the population. The disease will be spreading and the viral loads keep rising and the population will then be transferred to AIDS class. Some of the population will be removed through natural death rate (μ).

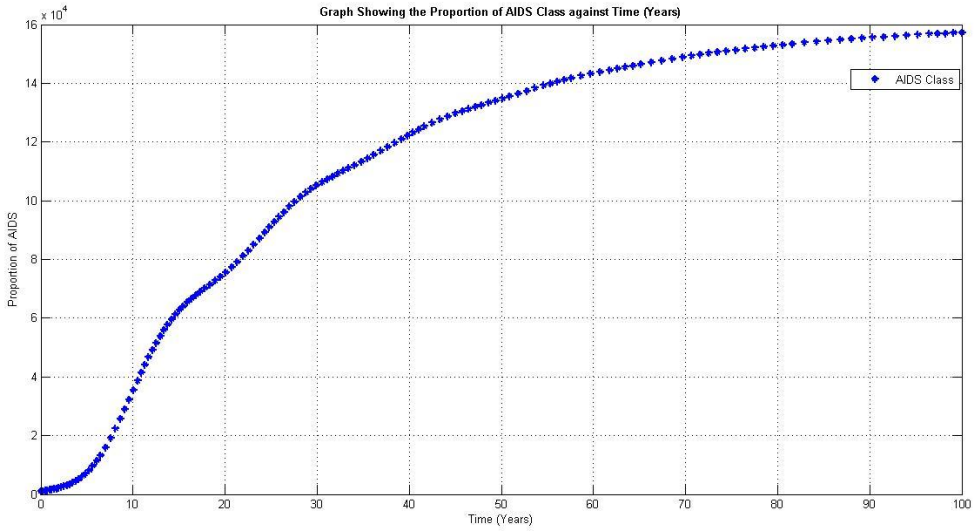

FIGURE 5.2.5: PROPORTION OF AIDS WITH TIME.

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Figure 5.2.5 shows individuals who are HIV/AIDS positive and are not on any therapeutic dose. It is found that these individuals initially increase without control. What this means is that AIDS patients without therapy will defiantly increase their viral load and all the opportunistic infections are bound to occur and the possibility of HIV/AIDS related death are inevitable. Those in the class will be removed from the population through HIV/AIDS related death (α) and any other natural related death (μ).

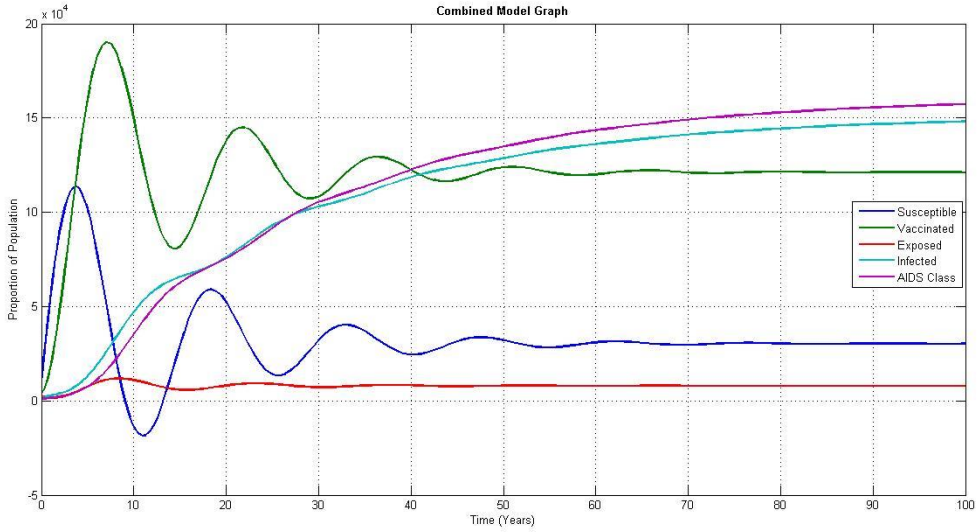

FIGURE 5.2.6: GENERAL VARIATION OF IN DIFFERENT CLASSES.

Figure 5.2.6 show variation of S, V, E, I and A with time when basic reproduction number R_{02} is greater than unity with the parameter values $\pi = 50,000,\ \delta_1 = 0.013,\ \rho = 0.025,$ $\mu = 0.0001,\ \theta_1 = 0.50,\ \phi = 07230,\ \tau = 0.987,\ c = 5.00,\ \alpha = 0.45$. It is observed that the susceptible and the vaccinated individuals' increases with time after attaining an equilibrium position decreases because of waning effect of the vaccine in protecting the individuals from infection. The exposed individuals increase slowly and remain stable. The infected and AIDS population increases slowly and finally rises without control indicating the presence of the infection and is infecting others who fall victim to it with the parameters value $\pi = 50,000,\ \delta_1 = 0.013,\ \rho = 0.025,$ $\mu = 0.0001,\ \theta_1 = 0.50,\ \phi = 07230,\ \tau = 0.987,\ c = 5.00,\ \alpha = 0.45$

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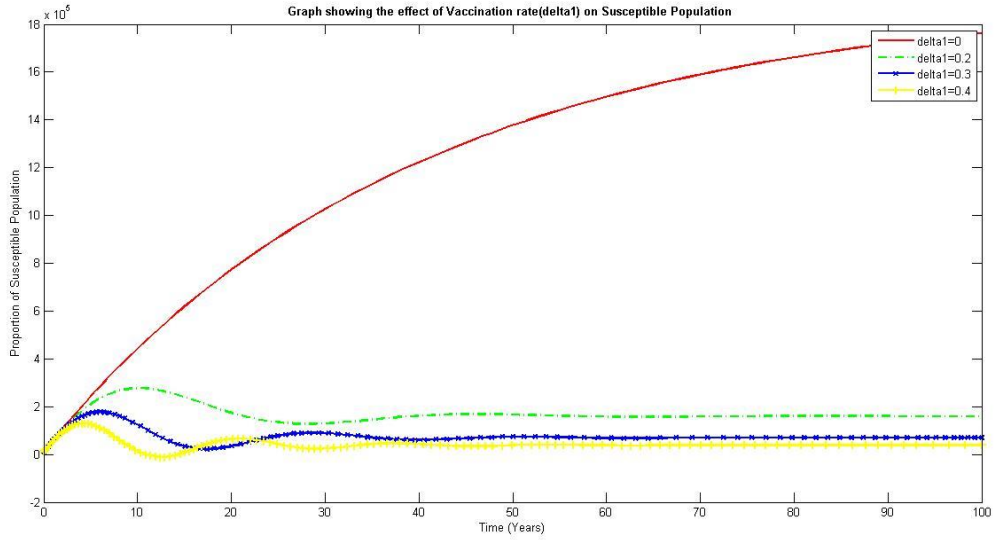

FIGURE 5.2.7: PROPORTION OF VACCINATION RATE ON THE SUSCEPTIBLE

Figure 5.2.7 shows the variation of the susceptible at different values of vaccination rate $\delta = 0.0,\ \delta = 0.2,\ \delta = 0.3,\ \delta = 0.4$. It is seen that as the vaccination rate increase, the rate at which the population becomes susceptible to the virus decrease with time. This means that the vaccine is very efficacious with very high potency. But after a long time the susceptible population will now move to the vaccination class since the vaccine has perfect efficacy.

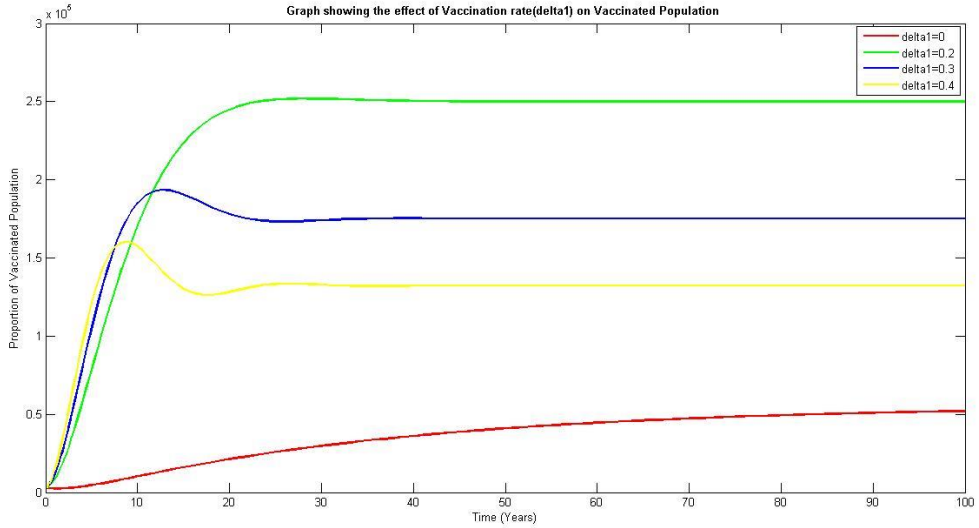

FIGURE 5.2.8: VARIATION OF VACCINATED WITH DIFFERENT VALUES OF VACCINATION RATE.

Figure 5.2.8 shows the variation of the vaccinated class with varying vaccinated rates. As seen from the graph, increase in vaccination rate will increase the number of people within the vaccinated population and in turn lead to

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high level of protection on the general population which may be exposed to the infection. However the waning rate of the vaccine may lead to some of these individual progresses to exposed class which may be exposed to infection. The only way an individual can be removed from the population is through the natural death rate (μ).

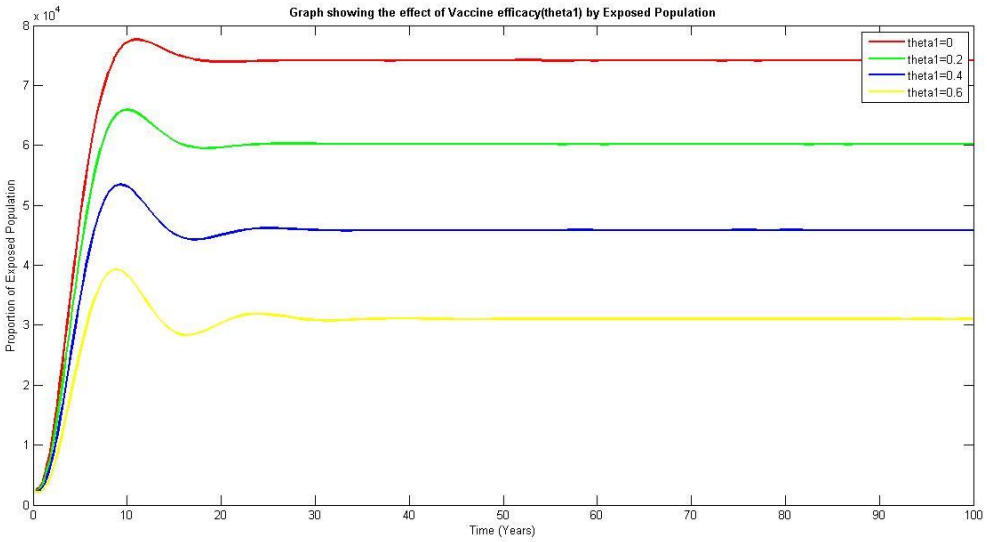

FIGURE 5.2.9: VARIATION OF THE EXPOSED WITH DIFFERENT VALUE OF VACCINE EFFICACY.

Figure 5.2.9 shows the variation of the exposed population with different values of vaccine efficacy rate $\theta = 0.0,\ \theta = 0.2,\ \theta = 0.4,\ \theta = 0.6$ with time, keeping other parameters constant. It is seen that as the vaccine efficacy increase the exposed population decreased, indicating that with good public health campaign for the population to receive compulsory vaccination against the virus, those that will be exposed to the virus will be highly reduce but not to zero. This may be because some may be exposed to at birth. Some fractions of the population may be removed through natural death and the waning rate of the vaccine transferred some fraction to infected class after being exposed to the virus.

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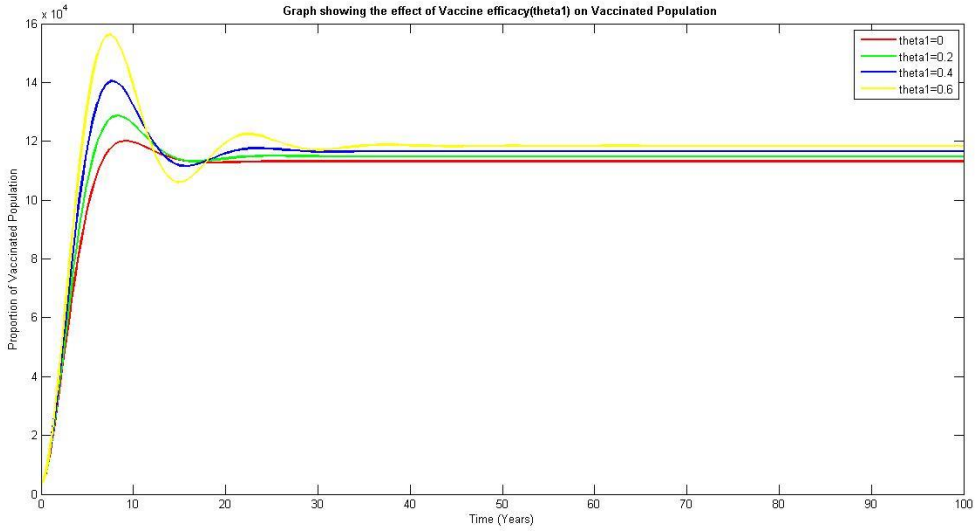

FIGURE 5.2.10: PROPORTION OF THE VACCINATED WITH DIFFERENT VALUES OF VACCINE EFFICACY.

Figure 5.2.9 shows the variation of the vaccinated individual population with different values of vaccine efficacy rates $\theta = 0.0,\ \theta = 0.2,\ \theta = 0.4,\ \theta = 0.6$ with time, keeping other parameters constant. It is seen that as the efficacy of the vaccine increases the vaccinated population also increases until the vaccine began to wane and decrease in the vaccinated population occurs after about 25 years, the population attain an equilibrium position and remain stable for a long time. The waning of the vaccine move some individual into an exposed population, making them susceptible to infection peradventures they have any slight contact with an infective. The removal rate from population is through the natural death with the parameters (μ).

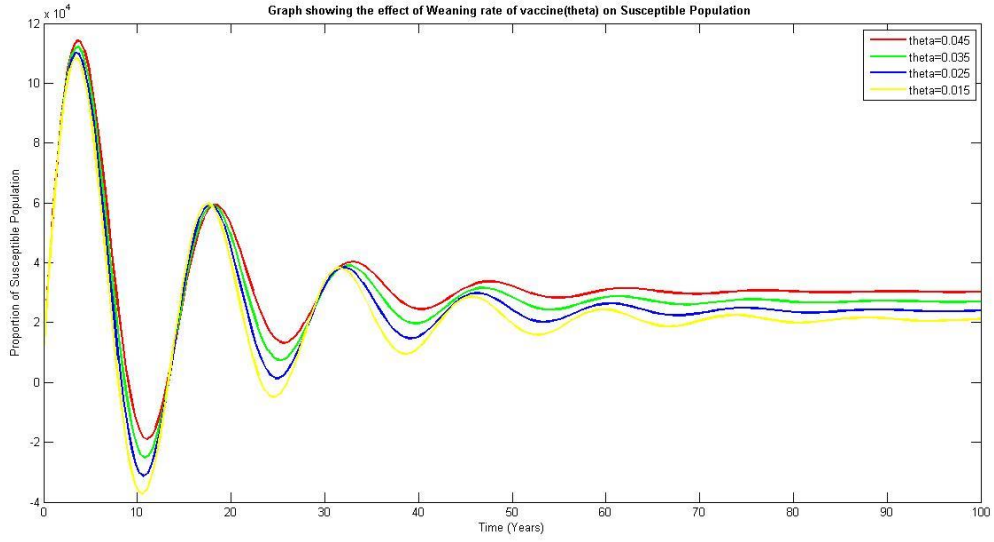

FIGURE 5.2.11: VARIATION OF THE SUSCEPTIBLE WITH DIFFERENT VALUES OF THE WANNING RATES OF THE VACCINE.

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Figure 5.2.11 shows the effect of different values of waning rates on the susceptible population. It is seen that if the vaccine wane the vaccinated population will become susceptible to the infection again. The vaccine is imperfect to protect the entire population from the effect of the virus and the disease will be maintained within the population using the sets of values for the waning rates $\theta = 0.045,\ \theta = 0.035,\ \theta = 0.025,\ \theta = 0.015$ keeping other parameters $\pi = 50,000,\ \delta_1 = 0.013,\ \rho = 0.025,\ \mu = 0.0001,\ \theta_1 = 0.50,\ \phi = 07230,\ \tau = 0.987,\ c = 5.00,\ \alpha = 0.45$ constant with time. It was discovered that, there was an increase in the susceptible population. The decrease is due to the waning effect of the vaccine not reading zero level due to the fact that vaccine will not wane totally. Vaccine alone may not be able to totally control the infection; combination with other control strategy may be needed.

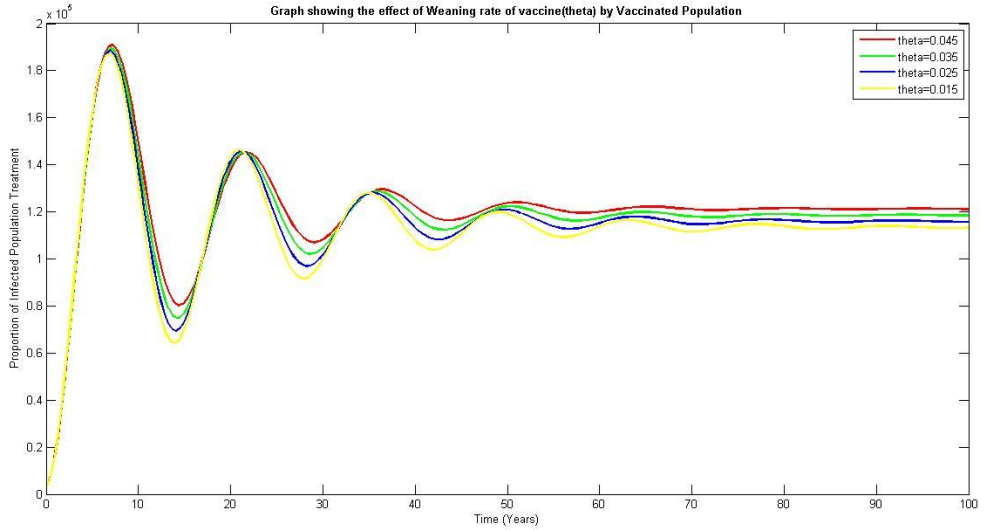

FIGURE 5.2.12:VARIATION OF THE VACCINATED WITH DIFFERENT VALUES OF WANNING RATE.

Figure 5.2.12 shows the variation in the vaccinated population with different values of waning rates $\theta = 0.045,\ \theta = 0.035,\ \theta = 0.025,\ \theta = 0.015$ with other parameters constant. It is seen that if the vaccine wane its potency, some individuals will leave the vaccinated class to the exposed class and may be infected with the virus if they have any contact with any infectious individual, meaning that the infection will keep rising within the population. As seen from the graph there is stable position after a long period indicating that vaccine alone cannot protect the population from the infection, a combination control strategies is needed to curbs the spread of the diseases.

11 CONCLUSION

Vaccine has the ability to combat HIV/AIDS and built it effect on the population. If the vaccine of 100 perfect efficacious. The current trial vaccine does not have 100% efficacy t3herefore should be treated with care. With effective public health campaign, individual should be encouraged to go for vaccination since even other vaccine the curbs the effect and spread of some disease are not 100% efficacious. Therefore, it would be advisable that the trial vaccine should be made public. HIV/AIDS centre for sex and vaccination should be established across the countries and the vaccination should be free. This will reduced the number of people that will be exposed to the disease and eventually reduce the infected rate. It was discovered that increase in vaccination rate eventually reduces the basic reproduction number R_0, meaning that few people will be infected.

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