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Mathematical Recipe for Curbing Human Papillomavirus Transmission Dynamics

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Abstract

A mathematical model for the transmission dynamics and control of Human Papillomavirus (HPV) was developed incorporating the impact of vaccination and condom usage. The effective reproduction number (R_c) was obtained and used to find the best recipe for curbing transmission of the disease. Using Nigerian demographic data, numerical simulations revealed that 20% HPV vaccination coverage of sexually active individuals is better than 75% condom usage on limiting the spread of HPV. Furthermore, it revealed that vaccinating 30% of individuals who are sexually active is a better way of curbing the disease than vaccinating 75% of individuals that are not yet sexually active.

1. Introduction

Human Papillomavirus (HPV) is a virus that affects the skin and the moist membranes that line the body, such as the throat, mouth, feet, fingers, nails, anus and cervix. HPV infection has been identified as a definite human carcinogen for six types of cancer: cervix, penis, vulva, vagina, anus and oropharynx (including

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the base of the tongue and tonsils). Over 100 known types of the virus have been identified of which 40 infect the female genital tract. Of these, 15 high risk types cause approximately 95% of cervical cancers (Moore *et al.*, 2009). Other types classified as low risk cause low-grade lesions including 75% to 95% of genital warts (Sahasrabuddhe *et al.*, 2007). In HIV negative women, the majority of HPV infections are cleared within $8-10$ months following infection (Firnhaber *et al.*, 2009). However, some infections evade the immune system through a complicated cascade of events and become persistent.

A human Papillomavirus-induced disease, notably cervical cancer is the second most common cancer in women worldwide by age-standardized incidence rate. In 2008, there were estimated 529,000 new cases and 274,000 deaths due to cervical cancer worldwide. More than 85% of cervical cancer deaths are in developing countries, where it accounts for 13% of all female cancers. The virus is common throughout the world. Although, most infections with it cause no symptoms, persistent genital HPV infection can cause cervical cancer in women. Virtually all cervical cancer cases (99%) are linked to genital infection with HPV which is the most common viral infection of the reproductive tract. The virus can also cause other types of anogenital cancer, head and neck cancers, and genital wart in both men and women.

At present, there are two HPV vaccines that are licenced, a bivalent (Cervarix) and a quadrivalent (Gardasil) HPV vaccine. Both vaccines are prophylactic (to prevent infection and subsequent disease). Therefore, public health workers target girls before they began having sex. The vaccines have been part of routine immunisation programmes in many wealthier countries since 2007. Nigeria is the most populous country in sub-Saharan Africa with more than 50 million sexually active females who are at risk of developing cervical cancer. Although most cancer cases in Nigeria are not documented, there is an estimated 14089 women diagnosed with cervical cancer and 8240 die from the disease annually. Furthermore, about 3.5% of women in the general population are estimated to harbour cervical HPV-16/18 infection at a given time, and 66.9% of invasive cervical cancers are attributed to HPVs 16 or 18 (Bruni *et al.*, 2015). Nigeria has not adopted national cervical cancer prevention policy nor included HPV vaccine in its immunization program (Morhason-Bello, 2013).

During the last 15 years Froelich *et al.* (2002), Dasbach *et al.* (2006), Al-arydad and Smith (2010), Laureen *et al.* (2012), Shernita and Ana (2012), Lee and Tanmeru (2012) and Francisco and Gonzalez (2012) have designed mathematical models of HPV transmission and control for different part of the world. Considering the works of the aforementioned authors, a new mathematical model was developed to complement and extend on their works by incorporating permanently HPV-infected compartment, vaccination and condom usage enhanced by public enlightenment campaign, standard incidence function and disease induced

death. These factors are very important in the transmission and control of human papillomavirus especially in developing countries where the disease is endemic.

2. Materials and Methods

2.1 Model Development

The Total population (N) is divided into seven (7) compartments of individuals not yet Sexually active (S_1), Susceptible individual (S_2), Vaccinated susceptible/recovered individuals (V_1), HPV infected individuals (H_1), Temporary Recovered individuals (R), Individuals permanently infected with one or more HPV types (H_2) and Vaccinated infected individuals (V_2). In the construction of the model we assumed that:

i. Individuals have equal chance of infection by the infectious individual in a case of a sexual contact.
ii. Public enlightenment campaign enhances HPV vaccination coverage and condom usage.
iii. Individuals under the ages of 15 years are not yet sexually active.
iv. HPV vaccine for all gender and age groups.

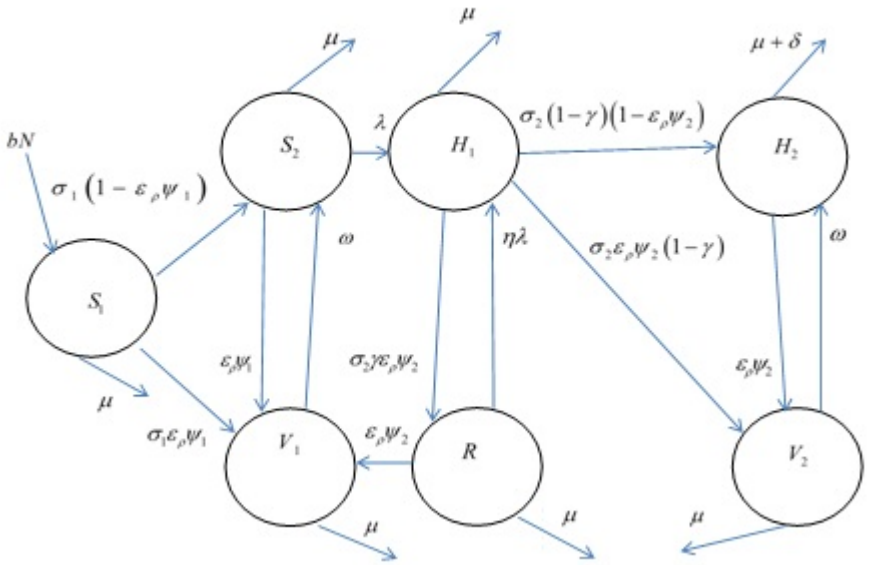

Figure 1: Schematic Diagram of HPV Transmission Dynamics and Control

The S_1 compartment represents individuals under the ages of 15 years and assumed not yet sexually active. This class is generated from the daily recruitment of individuals through birth at the rate b. They become sexually active and leave

the compartment to either S_2 or V_1 compartment at the rate σ_1. The parameters ε_ρ and ψ_1 are the efficacy of HPV vaccine and proportion of individuals not yet sexually active that received HPV vaccine which is enhanced by public enlightenment respectively. Thus, $\varepsilon_\rho\psi_1$ is the proportion of individuals who are not yet sexually active and received the vaccine, so that they are temporary immune from the disease and the term $(1-\varepsilon_\rho\psi_1)$ is the proportion of those not vaccinated.
The S_2 compartment represents the at-risk individuals that are prone to the disease. This class is generated from S_1 as explained above. They acquired infection and move to H_1 class through sexual transmission from individuals in the H_1, H_2 and V_2 compartments, given by the term $\frac{av\beta(H_1+H_2+V_2)(1-\epsilon_C\psi_C)}{N}$. The parameters a, v and β are the average number of sexual contact, average number of sexual partner(s) and HPV sexual transmission probability respectively, so that $av\beta$ is the effective sexual contact rate (rate that leads to infection in the absence of any control measure). The term $(1-\epsilon_c\psi_c)$ reflects the impact of condom usage on sexual transmission which is enhanced by public enlightenment campaign. The parameter (ϵ_c) is the condom efficacy rate and ψ_c is the proportion of individuals that uses condom. The compartment is increased due to waning off of vaccine efficacy from V_1 compartment at the rate of ω.
The V_1 compartment represents those who are vaccinated and immune within the life span of the vaccine. This class is generated from S_1 as earlier explained. They are increased from S_2 and R compartments due to effective vaccination of individuals in the compartments given by the term $\varepsilon_\rho\psi_2$, where ψ_2 is the proportion of individuals who are sexually active that received HPV vaccine which is enhanced by public enlightenment, so that $\varepsilon_\rho\psi_2$ is the proportion of individuals who are sexually active and received the vaccine. Furthermore, the class increases from H_1 by the term $\sigma_2\gamma\varepsilon_\rho\psi_2$, where σ_2 is the rate (average duration) at which individuals in H_1 must leave the compartment and γ is the proportion of individuals that recovered naturally from HPV, so that $\sigma_2\gamma\epsilon_\rho\psi_2$ is the proportion of sexually active individuals that recovered naturally and are vaccinated. The V_1 compartment diminishes to S_2 class due to waning off of vaccine at the rate ω.
The H_1 compartment represents those who are infected with HPV and can transmit the virus. This class is generated due to the effective sexual contact between $H1, H_2$ and V_2 with S_2 and R given by the terms $\frac{av\beta(H_1+H_2+V_2)(1-\epsilon_c\psi_c)}{N}$ and $\frac{av\beta\eta(H_1+H_2+V_2)(1-\varepsilon_c\psi_c)}{N}$ respectively. η is a modification parameter associated with reduced HPV sexual transmission probability (β) by recovered individuals. Individuals in the H_1 class leave the compartment at the rate σ_2 regardless of natural recovery/vaccination or neither. The parameters γ, ε_ρ and ψ_2 are as previously explained. And thus, $(1-\gamma)(1-\varepsilon_\rho\psi_2)$ is the proportion of those that did not recovered and did not received HPV vaccine respectively.
The R compartment represents those who recovered from HPV through their immune response and can be re-infected by the same or different type(s) of HPV if

their immune response is compromise. This class is generated from H_1 individuals that recovered naturally and not vaccinated given by the term $\sigma_2\gamma(1-\varepsilon_\rho\psi_2)$. They are decreased by effective sexual contact as explained above. Furthermore, the compartment reduces to V_1 class through vaccination given by the term $\epsilon_\rho\psi_2$. The H_2 compartment represents individuals who are having one or more HPV types permanently in their body. This class is generated from H_1 compartment due to lack of vaccination and natural recovery given by the term $\sigma_2(1-\varepsilon_\rho\psi_2)(1-\gamma)$. It is decreased to V_2 compartment due to effective vaccination $\varepsilon_\rho\psi_2$. It further decreases due to HPV-induced death at the rate δ, and increases due to waning off of vaccine of individuals in V_2 at the rate ω.

Lastly, the V_2 compartment represents those who are having one or more types of HPV in their body permanently but are vaccinated against the other HPV types. This class is generated from vaccinated individuals of H_1 and H_2 as explained earlier and diminishes to H_2 class due to waning off of the vaccine efficacy at the rate ω. Natural death occurs in all compartments at the rate μ.

The corresponding mathematical equations of the schematic diagram can be described by a system of ordinary differential equations given below:

$$\frac{dS_1}{dt} = bN - (\sigma_1 + \mu)S_1 \tag{1}$$

$$\frac{dS_2}{dt} = \sigma_1(1-\varepsilon_\rho\psi_1)S_1 - \frac{av\beta(H_1+H_2+V_2)(1-\varepsilon_c\psi_c)S_2}{N} + \omega V_1 - (\varepsilon_\rho\psi_2+\mu)S_2 \tag{2}$$

$$\frac{dV_1}{dt} = \sigma_1\varepsilon_\rho\psi_1 S_1 + \varepsilon_\rho\psi_2(S_2 + \sigma_2\gamma H_1 + R) - (\omega+\mu)V_1 \tag{3}$$

$$\frac{dH_1}{dt} = \frac{av\beta(H_1+H_2+V_2)(1-\varepsilon_c\psi_c)(S_2+\eta R)}{N} - (\sigma_2+\mu)H_1 \tag{4}$$

$$\frac{dR}{dt} = \sigma_2\gamma(1-\varepsilon_\rho\psi_2)H_1 - \frac{\eta av\beta(H_1+H_2+V_2)(1-\varepsilon_v\psi_c)R}{N} - (\varepsilon_\rho\psi_2+\mu)R \tag{5}$$

$$\frac{dH_2}{dt} = \sigma_2(1-\varepsilon_\rho\psi_2)(1-\gamma)H_1 + \omega V_2 - (\varepsilon_\rho\psi_2+\mu+\delta)H_2 \tag{6}$$

$$\frac{dV_2}{dt} = \sigma_2\varepsilon_\rho\psi_2(1-\gamma)H_1 + \varepsilon_\rho\psi_2 H_2 - (\omega+\mu)V_2 \tag{7}$$

where

$$N = S_1 + S_2 + V_1 + H_1 + R + H_2 + V_2 \tag{8}$$

so that

$$\frac{dN}{dt} = (b-\mu)N - \delta H_2 \tag{9}$$

in the biological-feasible region:

(10) $\Omega = \{(S_1, S_2, V_1, H_1, R, H_2, V_2) \in \mathbb{R}^7_+ : S_1+S_2+V_1+H_1+R+H_2+V_2 = N\}$

Let

$$\begin{gathered} \rho_1 = \varepsilon_\rho \psi_1 \\ \rho_2 = \varepsilon_\rho \psi_2 \\ \phi = \varepsilon_c \psi_c \\ K_1 = (\sigma_1 + \mu) \\ K_2 = (\rho_2 + \mu) \\ K_3 = (\omega + \mu) \\ K_4 = (\sigma_2 + \mu) \\ K_5 = (\rho + \mu + \delta) \end{gathered} \tag{11}$$

Here, (3.1) - (3.6 gives:)

$$\frac{dS_1}{dt} = bN - K_1 S_1 \tag{12}$$

$$\frac{dS_2}{dt} = \sigma_1(1-\rho_1)S_1 - \frac{av\beta(H_1+H_2+V_2)(1-\phi)S_2}{N} + \omega V_1 - K_2 S_2 \tag{13}$$

$$\frac{dV_1}{dt} = \sigma_1\rho_1 S_1 + \rho_2(S_2 + \sigma_2\gamma H_1 + R) - K_3 V_1 \tag{14}$$

$$\frac{dH_1}{dt} = \frac{av\beta(H_1+H_2+V_2)(1-\phi)(S_2+\eta R)}{N} - K_4 H_1 \tag{15}$$

$$\frac{dR}{dt} = \sigma_2\gamma(1-\rho_2)H_1 - \frac{\eta av\beta(H_1+H_2+V_2)(1-\phi)R}{N} - K_2 R \tag{16}$$

$$\frac{dH_2}{dt} = \sigma_2(1-\rho_2)(1-\gamma)H_1 + \omega V_2 - K_5 H_2 \tag{17}$$

$$\frac{dV_2}{dt} = \sigma_2\rho_2(1-\gamma)H_1 + \rho_2 H_2 - K_3 V_2 \tag{18}$$

2. Effective Reproduction Number, R_c

One of the most important concerns about any infectious disease is its ability to invade a population. The basic reproduction number, R_0 is a measure of the potential for disease spread in a population, and is inarguably one of the foremost and most valuable ideas that mathematical thinking has brought to epidemic theory (Heesterbeek and Dietz, 1996). It represents the average number of secondary cases generated by an infected individual if introduced into a susceptible population with no immunity to the disease in the absence of interventions to control the infection. If $R_0 < 1$, then on average, an infected individual produces less than one newly infected individual over the course of its infection period. In this case, the infection may die out in the long run. Conversely, if $R_0 > 1$, each infected individual produces, on average more than one new infection, the infection will be able to spread in a population. A large value of R_0 may indicate the possibility of a major epidemic. Similarly, the effective reproduction number, R_c represent the average number of secondary cases generated by an infected individual if introduced into a susceptible population where control strategies are used.

Using the next generation operator technique described by Diekmann and Heesterbek (2000) and subsequently analysed by Van de Driesches and Watmough (2002), we obtained the effective reproduction number, R_c of our model which is the spectral radius of the next generation matrix, K, where $K = FV^{-1}$. The matrices of F (for the new infection terms) and V (of the transition terms) are obtained from the infected compartment (i.e. H_1, H_2 and V_2) at disease-free equilibrium (E^0).

Now, the disease-free equilibrium of the model exist at the point

$$E^0 = \begin{pmatrix} S_1^0 \\ S_2^0 \\ V_1^0 \\ H_1^0 \\ R^0 \\ H_2^0 \\ V_1^0 \end{pmatrix} = \begin{pmatrix} \frac{bN^0}{K_1} \\ \frac{\sigma_1 b(K_3(1-\rho_1)+\omega\rho_1)N^0}{K_1(K_2K_3-\omega\rho_2)} \\ \frac{\sigma_1 b\{(\rho_1(K_2K_3-\omega\rho_2))+\rho_2(K_3(1-\rho_1)+\omega\rho_1)\}N^0}{K_1K_3(K_2K_3-\omega\rho_2)} \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \tag{19}$$

and thus we have,

$$F = \begin{pmatrix} \frac{av\beta(1-\phi)S_2^0}{N^0} & \frac{av\beta(1-\phi)S_2^0}{N^0} & \frac{av\beta(1-\phi)S_2^0}{N^0} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \tag{20}$$

and

$$V^{-1} = \begin{pmatrix} \frac{1}{K_4} & 0 & 0 \\ \frac{\sigma_2(1-\gamma)(\omega\rho_2+K_3(1-\rho_2))}{K_4(K_3K_5-\omega\rho_2)} & \frac{K_3}{(K_3K_5-\omega\rho_2)} & \frac{\omega}{(K_3K_5-\omega\rho_2)} \\ \frac{\sigma_2\rho_2(1-\gamma)(K_5+(1-\rho_2))}{K_4(K_3K_5-\omega\rho_2)} & \frac{\rho_2}{(K_3K_5-\omega\rho_2)} & \frac{K_5}{(K_3K_5-\omega\rho_2)} \end{pmatrix} \tag{21}$$

The effective reproduction number is then given as:

$$R_c = \frac{av\beta(1-\phi)[K_3K_5 - \omega\rho_2 + \sigma(1-\gamma)(\omega\rho_2 + K_3(1-\rho_2)) + \sigma\rho_2(1-\gamma)(K_5 + (1-\rho_2))]S_2^0}{K_4(K_3K_5 - \omega\rho_2)N^0} \tag{22}$$

3. Results and Discussions

3.1. **Estimation of Variables and Population-dependent Paramaters Values.** Variables and population-dependent parameters of a model depend on the disease epidemiology and the demographic profile of the population (country) concerned. Though, the model can fit into any population set up, we used Nigerian demographic profiles.

The total population of Nigeria is given as 181,562,056 in year 2015 with 78,083,541 under fifteen (15) years of age and 103,478,515 to be fifteen (15) years and above (CIA, 2016). As no any single research work (to the best of our knowledge) is available on HPV prevalence in the entire population of Nigeria, we analyze recent data of HPV seroprevalence research works carried out by different authors at different parts of the country. The authors are Okolo et al. (2010), Piras *et al.* (2011), Auwal *et al.* (2013), Nweke *et al.* (2013), Musa *et al.* (2013), Fadahunsi *et al.* (2013) and Akarolo-Anthony *et al.* (2014). Though, all the researchers focus on women seroprevalence, we assumed the same rate for men since the research carried out by Smith *et al.* (2011) with 14,800 men in 23 countries showed that anogenital HPV DNA prevalence is generally high is sexually active men, ranging from 1% to 84% in low risk men and 2% to 93% in high risk men. The results of the analysis are presented on Table 1. Due to inadequate information and high cost of HPV vaccine, many individuals have not been vaccinated. Thus, we assumed 100,000 individuals each for V_1 and V_2.

Population-dependent parameters of the model are the birth rate (b), the death rate (μ) and the average number of sexual contacts (a). The birth rate and life expectancy of Nigerians are given as 37.64 births per 1000 per year and 53.02 years respectively (CIA, 2016). Thus, the birth rate is 0.038 per year and the death rate is equal to $\frac{1}{53.02}$, that is 0.019 per year. The average number of sexual contacts for Nigerians is estimated to be 10 per year. It is important to note that this is the average number of sexual contacts of all sexually active individuals in the country and not just an individual.

3.2. **Estimation of Population-independent Parameters Values.** This data are estimated based on HPV epidemiology and published data. In a study conducted by Burshell *et al.* (2015), they found out that if one person in a heterosexual couple has HPV, there is a 20% chance his/her partner will pick up the virus in six months. That is 20% of half a year. This resulted to $\beta = 0.4$. And due to reduced sexual transmission rate of R, it is assumed that $\eta = 0.75$ so that $\eta\beta = 0.3$. Furthermore, the efficacy of HPV vaccine and condom has been reported to be 0.95 (CDC, 2015) and 0.85 (Holmes *et al.*, 2004) respectively.
So far studies have shown people to still be protected after 9 years for Gardasil and 6 years for Cervarix, the duration of immunity of the two HPV vaccines is not yet ascertained. But with passage of time and longer experience, vaccine protection against most diseases has been shown for at least 25 years in those who showed an adequate initial response to the primary course of vaccinations (Van Damme and Van Herek, 2007). Thus, we have the average waning rate of HPV vaccine (ω) assumed to be 25 years. That is, 0.04 per year as in most vaccines. About 70% of individuals infected with HPV cleared the disease within a year and about 20% cleared the disease within 2 years (Myers *et al,* 2000). Thus we have $\sigma_2 = \frac{70}{100} + \left(\frac{20}{100} \times \frac{1}{2}\right)$, that is, 0.8 per year. Similarly, individuals in S_1 compartment leaves the class after fifteen (15) years on average, thus the rate of becoming sexually active (σ_1) is equal to 0.067 per year.
The proportion of individuals that recovered from HPV infection is generally agreed to be 90%, which is $\gamma = 0.9$. The HPV-induced death (δ) is estimated to be 0.0027. And lastly, the control parameters ψ_1, ψ_2 and ψ_3 ranges between 0 and 1, while average number of sexual partner(s) (V) is greater than zero. Table 1, summarizes the results above.

S/N	**Variable/Parameter**	**Value**	**S/N**	**variable/Parameter**	**value**
1	S_1	78,083,541	12	β	0.4
2	S_2	38,518,181	13	η	0.75
3	V_1	100,000	14	ε_ρ	0.9
4	H_1	29,187,150	15	ε_c	0.75
5	R	32,430,167	16	ω	0.04
6	H_2	3,143,017	17	σ_1	0.067
7	V_2	100,000	18	σ_2	0.8
8	N	181,562,056	19	γ	0.9
9	b	0.038	20	β	δ
10	μ	0.019	21	ψ_1, ψ_2, ψ_c	(0.1)
11	a	10	22	v	> 0

Table 1: Variables and Parameters values of the model

3.3. **Numerical Simulations.**

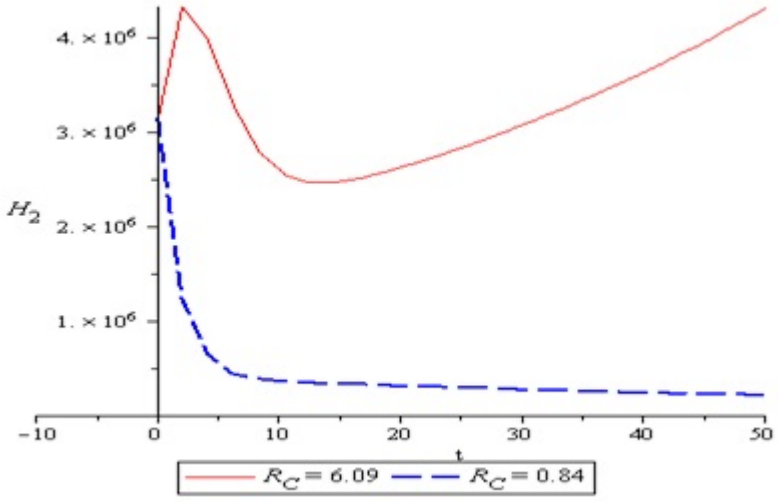

Figure 2: The effect of effective reproduction number on the morbidity of permanently infected individuals with one or more HPV strain(s), H_2. Initial variables and parameters value used are as in Table 1 with $\psi_1 = \psi_2 = \psi_c = 0.4$ $v = 2$, and $\psi_1 = \psi_2 = \psi_c = 0.75, v = 1$ which gives $R_c = 6.09$ and $R_c = 0.84$ respectively.

Figure 1 shows the persistence of the disease when $R_c = 6.09$ and convergence to disease free when $R_c = 0.84$. This is in agreement with the conditions of the reproduction number that whenever $R_c \leq 1$ the disease will be wipe out from the population sooner or later and if $R_c > 1$ the disease will persist leading to endemic situation of the disease. Furthermore it revealed that with 40% vaccination coverage each for both individuals not yet sexually active and those that are sexually active, 40% of condom usage and an average number of 2 sexual partners the disease will still persist in the country. A necessary recipe to reduce the menace of the disease in the country is for 75% of vaccination coverage each for both individuals not yet sexually active and those that are sexually active as well as 75% condom usage with an average number of 1 sexual partner.

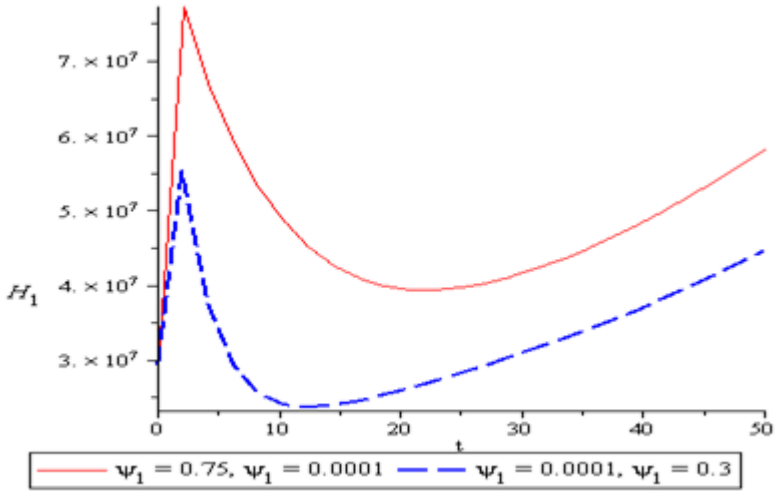

Figure 3: A Comparison between the effect of HPV vaccine on the morbidity of individuals before and when sexually active on the morbidity of H_1 individuals. Initial variables and parameters value used are as in Table 1 with $\psi_1 = 0.75, \psi_2 = 0.0001, \psi_c = 0.01, v = 4$ and $\psi_1 = 0.0001, \psi_2 = 0.2, \psi_c = 0.01, v = 4$ which gives $R_c = 108.8$ and $R_c = 36.34$ respectively.

Figure 2 compares the effect of vaccination before been sexually active and when sexually active. It clearly revealed that vaccinating 30% of sexually active individuals resulted to less number of HPV infected individuals (H_1) compared to vaccinating 75% of individual not yet sexually active. This is so because it is the sexually active individuals that transmit the disease. Though, both alone do not lead to disease free situation in the long run. This is in partial conflict with the World Health Organisation and Centre for disease control that emphasis more on vaccinating individuals not yet sexually active, although, it may have to do with the lack of certainty of the vaccine safety on individuals with age greater than 26 years.

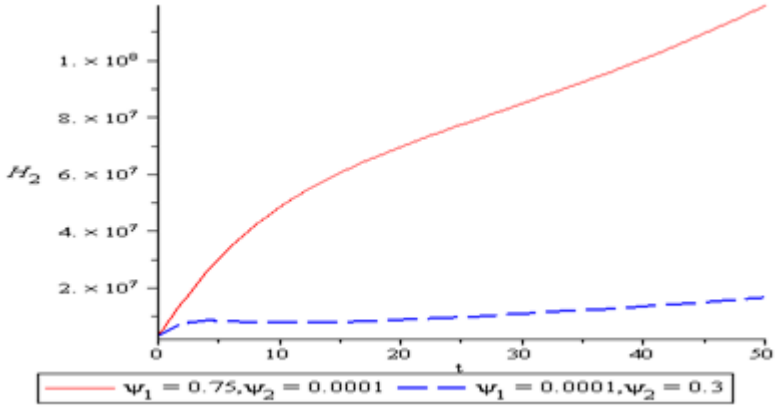

Figure 4: A Comparison between the effect of HPV vaccine when sexually active and before been sexually active on the morbidity of H_2 individuals. Initial variables and parameters value used are as in Figure 2 above.

Similarly, as in figure 2, the effect of 30% vaccination of sexually active individuals on individuals permanently with one or more HPV type(s) (H_2) is far better than 75% vaccination coverage of not yet sexually active individuals. Though, none of them brings about disease free situation.

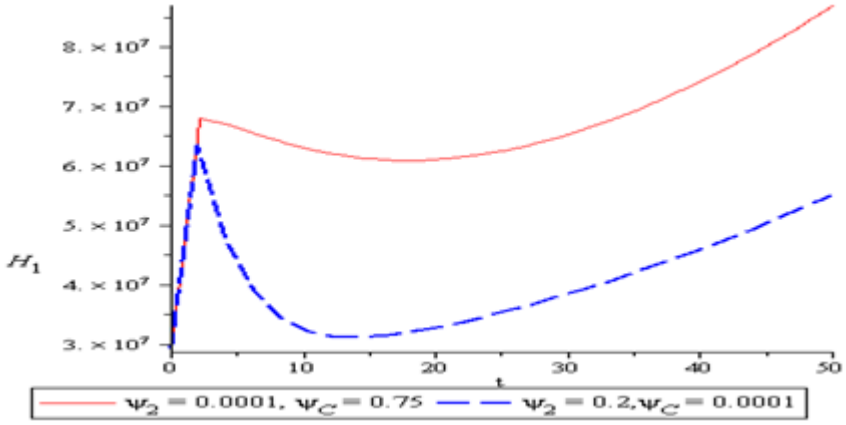

Figure 5: A Comparison between the effect of HPV vaccine given to sexually active individuals and condom usage on the morbidity of H_1 individuals. Initial variables and parameters value used are in Table 1 with $\psi_1 = 0.0001, \psi_2 = 0.0001, \psi_c = 0.75, v = 4$ and $\psi_1 = 0.0001, \psi_2 = 0.2, \psi_c = 0.0001, v = 4$ which gives $R_c = 51.64$ and $R_c = 36.34$ respectively.

Next is a comparison between the impact of HPV vaccine and condom usage both of sexually active individuals on the morbidity of H_1. Though, a typical condom

efficacy is 85%, meaning that 15 people out of every 100 will become pregnant or infected with sexually transmitted disease during the first year of usage, the impact of HPV vaccine with 95% efficacy on the transmission dynamics of the disease is higher as clearly showed on figure 4.

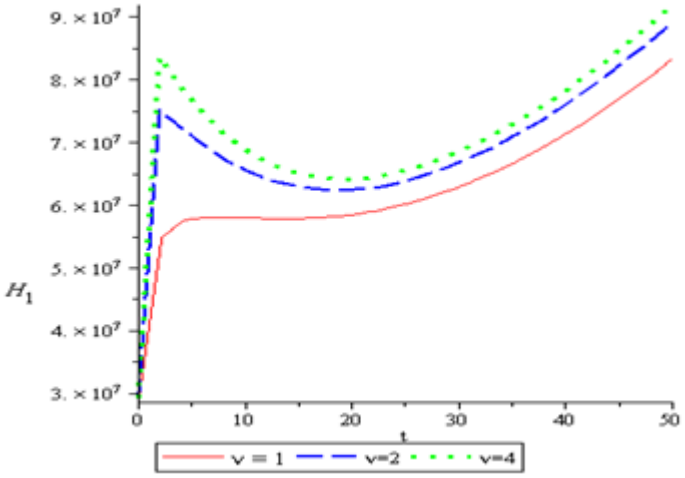

Figure 6: The effect of average number of sexual partner(s) on the morbidity of infected individuals with HPV, H_1. Initial variables and parameters value used are as in Table 1 with $\psi_1 = \psi_2 = 0.0001, \psi_c = 0.01$ and $v = 1, 2, 3$ which gives $R_c = 35.31, R_c = 70.61$ and $R_c = 141.24$ respectively.

In figure 5, we observed that higher number of sexual partners (v) have negative effect on the morbidity and hence mortality of HPV. Thus, The higher the number of sexual partners the higher the morbidity of H_1.

4. Conclusion

We developed a mathematical model as a recipe to curtail the transmission dynamics of Human Papillomavirus. The disease free equilibrium state (E^0) and effective reproduction number (R_c) was obtained. Our results showed that for the disease to be totally eradicated $R_c < 1$. This is hardly possible, as to vaccinate 75% of both sexually active and not yet sexually active individuals as well 75% condom usage is rarely possible in any population. Though, the recipe for the disease menace is to curb the disease to bearable minimum by vaccinating more of sexually active individuals.

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