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An Age-Structured Mathematical Model for Human Papillomavirus (HPV) and Cervical Cancer in the Presence of Vaccination and Treatment

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Abstract: This study develops an age-structured mathematical model to investigate the dynamics of Human Papillomavirus (HPV) and cervical cancer progression in the presence of vaccination and treatment. The model stratifies the population into five epidemiological classes across discrete age groups to capture differences in disease transmission and intervention outcomes. Analytical results establish conditions for the stability of the disease-free and endemic equilibria depending on the basic reproduction number (R_0). Numerical simulations show that early vaccination of adolescents significantly reduces HPV prevalence, while treatment improves outcomes in older populations. The combined effect of vaccination and treatment proves most effective, leading to projected reductions of over 70% in cervical cancer incidence within 25 years. The findings highlight the importance of integrated, age-targeted strategies for achieving cervical cancer elimination goals.

Keywords: *HPV; Cervical Cancer; Age-Structured Model; Vaccination; Treatment; Mathematical Epidemiology.*

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I. INTRODUCTION

Cervical cancer remains one of the leading causes of cancer-related mortality among women, particularly in low- and middle-income countries. Persistent infection with high-risk types of Human Papillomavirus (HPV) is the primary cause of cervical cancer [11]. In 2020, the International Agency for Research on Cancer reported more than 600,000 new cases and over 340,000 deaths worldwide, with nearly 90% of these occurring in low- and middle-income countries [11]. Despite progress in preventive vaccines and treatment options, cervical cancer continues to be the fourth leading cause of cancer-related deaths globally [12].

Vaccination against HPV has been shown to significantly reduce the incidence of HPV infections and precancerous lesions [1], [17]. Mathematical models demonstrate that achieving high vaccine coverage among adolescents provides long-term protection and herd immunity [3], [20]. However, vaccine uptake remains uneven across the world, especially in low-resource settings [6]

Treatment of precancerous and cancerous lesions is also vital, particularly for older cohorts already exposed to HPV [5]. However, treatment alone is insufficient to eradicate HPV because new infections continue to occur [9]. This underscores the need for integrated strategies that combine vaccination and treatment. Such combined approaches are aligned with the World Health Organization's goal of cervical cancer elimination by 2030 [19].

Age-structured modeling provides critical insights into HPV dynamics because transmission, vaccination effectiveness, and cancer progression vary across age groups [8], [15]. Apima and Mutwiwa (2023) [2] demonstrated the importance of incorporating vaccination into HPV models, showing that vaccine efficacy significantly alters disease dynamics. By building on their framework, this study integrates both vaccination and treatment into an age-structured model, addressing existing gaps in the literature.

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II. METHODOLOGY

➢ *Model Structure*

The total population is divided into five epidemiological classes: Susceptible individuals $S(a,t)$, Infected individuals $I(a,t)$, Individuals with persistent HPV infection progressing toward cancer $C(a,t)$, Vaccinated individuals $V(a,t)$ and Individuals receiving treatment $R(a,t)$

Here, a denotes age and t denotes time. The model considers natural recruitment into the susceptible class, transmission through effective contact, vaccination of susceptible individuals, progression from infection to cancer, and treatment of infected individuals [2], [5].

➢ *Parameters and Assumptions*

Key parameters include transmission rate (β), vaccination rate (η), treatment rate (δ), vaccine efficacy (ρ), cancer progression (γ), and mortality (v). Vaccination reduces susceptibility, treatment decreases mortality, and progression to cancer is more significant among older age groups [3], [6].

Table 1 Description of Variables and Parameter

Variables	**Description**
$S(a,t)$	The class of individuals susceptible to HPV and cervical cancer infections.
$I(a,t)$	Consists of individuals who are asymptomatically infected with HPV infection
$C(a,t)$	Class due to persistence of the HPV infection
$V(a,t)$	represent the class Vaccinated against HPV
$R(a,t)$	represent the class infected with HPV and receiving Treatment
Parameter	**Description**
μ	The rate natural death is assumed to occur in all classes
γ	is the rate of progression to cervical cancer
α	The rate at which Most HPV infected Individuals recovers from HPV infection and slide back to the $S(t)$ class
λ	Transmission rate from the susceptible compartment to the infection compartment
ρ	denotes the probability of the successes of the vaccination
v	The rate Mortality occurs among cervical cancer patients
Λ	The rate at which Recruitment into susceptible class is done
η	The rate at which Vaccination is done
δ	represent the rate at which individuals infected with HPV are receiving treatment
a	represent the age
t	represents the time

➢ *Force of Infection*

The probability of acquiring HPV infection depends on contact with infected individuals and vaccine efficacy [2].

$$\lambda = (1-\rho)\frac{k\beta I(a,t)}{N(a,t)} \quad (1)$$

Where $N(a,t)$ is the total population in age group a at time t.

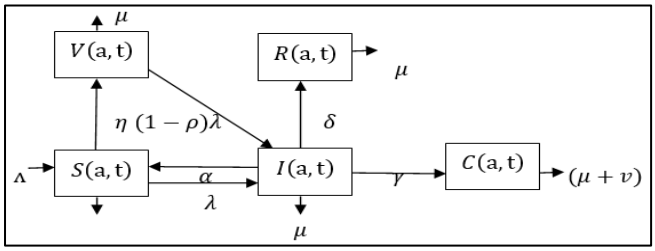

Fig 1 Flow Diagram of the HPV and Cervical Cancer Model in the Presence of Vaccine and Treatment.

➢ *Model Equations*

The dynamics are described by a system of PDEs [3], [8]

$$\frac{\partial S(a,t)}{\partial a} + \frac{\partial S(a,t)}{\partial t} = \Lambda(a) + \alpha(a)I(a,t) - \left(\eta(a) + (1-\rho)\frac{k(a)\beta(a)I(a,t)}{N(a,t)} + \mu(a)\right)S(a,t) \quad (2)$$

$$\frac{\partial I(a,t)}{\partial a} + \frac{\partial I(a,t)}{\partial t} = (1-\rho)\frac{k(a)\beta(a)I(a,t)}{N(a,t)}S(a,t) + (1-\rho)\frac{k(a)\beta(a)I(a,t)}{N(a,t)}V(a,t) - (\alpha(a) + \gamma(a) + \delta(a) + \mu(a))I(a,t) \quad (3)$$

$$\frac{\partial C(a,t)}{\partial a} + \frac{\partial C(a,t)}{\partial t} = \gamma(a)I(a,t) - (v(a) + \mu(a))C(a,t) \quad (4)$$

$$\frac{\partial V(a,t)}{\partial a} + \frac{\partial V(a,t)}{\partial t} = \eta(a)S(a,t) - (1-\rho)^2\frac{k(a)\beta(a)I(a,t)}{N(a,t)}V(a,t) - \mu(a)V(a,t) \quad (5)$$

$$\frac{\partial R(a,t)}{\partial a} + \frac{\partial R(a,t)}{\partial t} = \delta(a)I(a,t) - \mu(a)R(a,t) \quad (6)$$

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The total population $N(a,t)$ at a given age a and time t can be represented as the sum of individuals in each class:

$$\boldsymbol{N(a,t) = S(a,t) + I(a,t) + C(a,t) + V(a,t) + R(a,t)} \quad (7)$$

The PDEs are converted into ODEs using age discretization [15].

Let $S_i(t), I_i(t), C_i(t), V_i(t), R_i(t)$ represent the respective compartments for age group i, and let

$$N_i(t) = S_i(t) + I_i(t) + C_i(t) + V_i(t) + R_i(t) \quad (8)$$

Where $i = 1,2,3$, etc

The resulting ODEs for each age group are as follows:

$$\frac{\mathrm{dS_i(t)}}{\mathrm{dt}} = \Lambda_\mathrm{i} + \alpha_\mathrm{i} I_i(t) - \left[\eta_\mathrm{i} + (1-\rho)\frac{k_i \beta_\mathrm{i} I_i(t)}{N_i(t)} + \mu_\mathrm{i}\right] S_i(t) \quad (9)$$

$$\frac{dI_i(t)}{dt} = (1-\rho)\frac{k_i \beta_\mathrm{i} I_i(t)}{N_i(t)} S_i(t) + (1-\rho)\frac{k_i \beta_\mathrm{i} I_i(t)}{N_i(t)} V_i(t) - [\alpha_\mathrm{i} + \gamma_\mathrm{i} + \delta_\mathrm{i} + \mu_\mathrm{i}] I_i(t) \quad (10)$$

$$\frac{\mathrm{dC_i(t)}}{\mathrm{dt}} = \gamma_\mathrm{i} I_i(t) - [\mathrm{v_i} + \mu_\mathrm{i}]\mathrm{C_i(t)} \quad (11)$$

$$\frac{\mathrm{dV_i(t)}}{\mathrm{dt}} = \eta_\mathrm{i} S_i(t) - (1-\rho)^2 \frac{k_i \beta_\mathrm{i} I_i(t)}{N_i(t)} V_i(t) - \mu_\mathrm{i} V_i(t) \quad (12)$$

$$\frac{\mathrm{dR_i(t)}}{\mathrm{dt}} = \delta_\mathrm{i} I_i(t) - \mu_\mathrm{i}\mathrm{R_i(t)} \quad (13)$$

The population is divided into five discrete age groups: 8–12, 13–17, 18–24, 25–34, and 35+.

Table 2 Age Groups used

S/N	Age Group (years)	Description
1.	8–12	Early adolescence, the primary target for HPV vaccination campaigns.
2.	13–17 years	Adolescents, with some susceptibility to HPV due to early exposure.
3.	18–24 years	Young adults, representing the population at the highest risk of HPV infections.
4.	25–34 years	Adults transitioning into middle age with cumulative infection risks.
5.	35+ years	Older adults, where cancer progression and treatment outcomes are significant.

➢ *Reproduction Number and Stability Analysis*

Using the next-generation matrix approach [22], the basic reproduction number R_0 is derived as:

$$R_0 = \frac{\boldsymbol{\beta_\mathrm{i}\, \Lambda_\mathrm{i}}}{\boldsymbol{\mu_\mathrm{i}\,(\eta_\mathrm{i} + \mu_\mathrm{i})}}, \quad (14)$$

With $\mathrm{F} = \beta_\mathrm{i}\, \Lambda_\mathrm{i}$ and $V = \mu_\mathrm{i}\,(\eta_\mathrm{i} + \mu_\mathrm{i})$ where F is the new infection matrix, V is the transition matrix of infected classes. Analytical evaluation shows that if $R_0 < 1$, the DFE is globally asymptotically stable [22], and if $R_0 > 1$, the EE exists and is stable [3], [8].

III. RESULTS AND DISCUSSION

A. Stability of Equilibria

The system of equations defined in Section 2.4 governs the transition of individuals across compartments. At equilibrium, the derivatives vanish $\left(\frac{dS}{dt} = \frac{dI}{dt} = \frac{dC}{dt} = \frac{dV}{dt} = \frac{dR}{dt} = 0\right)$, yielding steady states.

For the disease-free equilibrium (DFE), we set $I_i(t) = C_i(t) = 0$, giving:

$$R* = 0. \quad V* = \frac{\eta_i}{\mu_i} S*, \quad S* = \frac{\Lambda_i}{\eta_i + \mu_i},$$

Thus the DFE is:

$$(S*,\ I*, *, V*, R*) = \left(\frac{\Lambda_i}{\eta_i + \mu_i}, 0, 0, \frac{\eta_i}{\mu_i} S*, 0\right) \quad (15)$$

If $\mathrm{R_0} < 1$, then $\frac{dI}{dt} = \lambda S - (\gamma + \delta + \mu) I$ is negative, meaning infection terms decay faster than recruitment and the DFE is asymptotically stable [22]. This corresponds to a population where HPV is absent but vaccination continues to shift susceptible into the vaccinated class. Linearization of the system around the DFE and application of the next-generation matrix shows that the dominant eigenvalue equals the basic reproduction number (R_0).

Conversely, when $R_0 > 1$, the system admits a positive endemic equilibrium (EE), which becomes stable [3], [8]. Then infection terms dominate, making the endemic equilibrium feasible, where $I* > 0$ and $C* > 0$.

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Thus, the Endemic Disease Equilibrium (EFE) is determined as:

$$E^* = \{S^*_{EE}, I^*_{EE}, C^*_{EE}, V^*_{EE}, R^*_{EE}\} \quad (16)$$

This explains why the persistence of HPV in the absence of sufficient vaccination/treatment is inevitable.

The DFE corresponds to a state where no infection persists. It is stable if $R_0 < 1$. The endemic equilibrium exists when $R_0 > 1$, indicating persistent transmission [3], [8].

B. Sensitivity Analysis

Sensitivity analysis helps identify which parameters have the most significant impact on the basic reproduction number R_0. This allows for a better understanding of how changes in key factors influence the spread of HPV and cervical cancer. The sensitivity index is calculated using the formula:

$$S_x^{R_o} = \frac{\partial R_0}{\partial x} X \frac{x}{R_0} \quad (17)$$

Where x represents the parameters affecting R_0. Using the equation (15) we compute the sensitivity indices for each parameter.

Sensitivity of R_0 to β_i (Transmission Rate) is

$$\frac{\partial R_0}{\partial \beta_i} = \frac{\Lambda_i}{\mu_i (\eta_i + \mu_i)} \quad (18)$$

Multiplying by $\frac{\beta_i}{R_0}$, the sensitivity index simplifies to:

$$S_{\beta_i}^{R_o} = 1 \quad (19)$$

This indicate that 10% increase in the transmission rate β_i, leads to a 10% increase in R_0, making it one of most influential parameters in HPV transmission.

Sensitivity of R_0 to Λ_i (Recruitment Rate)

$$\frac{\partial R_0}{\partial \Lambda_i} = \frac{\beta_i}{\mu_i (\eta_i + \mu_i)} \quad (20)$$

Thus, the sensitivity index is (28) Similar to β_i, a 10% increase in the recruitment rate (Λ_i) leads to a 10% increase in R_0, showing its strong effect on the infection levels.

Sensitivity of R_0 to μ_i (Natural death Rate)

$$\frac{\partial R_0}{\partial \mu_i} = -\frac{\beta_i(\eta_i + 2\mu_i)}{\mu_i^2 (\eta_i + \mu_i)^2} \quad (21)$$

This results in a negative sensitivity index, meaning that higher natural death rates lead to a reduction in R_0. A 10% increase in μ_i decreases R_0, indicating that shorter lifespan lower HPV transmission.

Sensitivity of R_0 to η_i (Progression Rate from Infection to Cancer)

$$\frac{\partial R_0}{\partial \eta_i} = -\frac{\beta_i \Lambda_i}{\mu_i (\eta_i + \mu_i)^2} \quad (22)$$

Since the sensitivity index is negative, this means that increasing the progression rate from HPV infection to cancer reduces R_0, likely due to faster progression limiting further transmission.

➢ *Interpretation of Sensitivity Analysis*

From the computed sensitivity indices, the transmission rate (β_i) and recruitment rate (Λ_i) have the highest positive impact on R_0. This means that interventions targeting reduced transmission (e.g., condom use, HPV awareness campaigns) and controlled population dynamics (e.g., vaccination at early ages) can significantly lower the spread of HPV.

On the other hand, the natural death rate (μ_i) and cancer progression rate (η_i) have a negative impact on R_0. Although this is not a desirable control technique, it implies that HPV transmission is limited by increased mortality and faster disease development. The sensitivity analysis shows that lowering transmission rates by behavioral interventions and vaccination, while assuring early treatment to avoid protracted infection periods, are the most effective approach for minimizing HPV transmission. Public health interventions should emphasize efforts to increase access to HPV treatment and vaccine coverage.

Sensitivity analysis shows β and λ increase R_0, while μ and γ reduce it [2], [5].

Table 3 Model Parameters and their Sources

Parameter	Description	Value	Source
Λ_i	Reflects the natural replenishment of the population.	0.02	WHO (2024)
k_i	Effective contact rate with HPV-infected individuals in group i. Represents the average number of contacts sufficient for HPV transmission per unit time.	0.8	Mutwiwa & Apima (2023)
β_i	Transmission rate of HPV per effective contact for individuals in group i.	0.5	Mutwiwa & Apima (2023)
η_i	Vaccination rate of susceptible individuals in group i.	0.03	Mutwiwa & Apima (2023)
μ_i	Natural death rate of individuals in group i.	0.01	WHO (2024)
v_i	Mortality rate among cervical cancer patients in group i.	0.10	WHO (2024)
ρ_i	Probability of vaccination success ($1 - \rho_i$ reflects	0.85	Assumed (high vaccine

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	vaccine efficacy for group i).		coverage scenarios)
γ_i	Progression rate from HPV infection to cervical cancer in group i.	0.02	Mutwiwa & Apima (2023)
δ_i	Treatment rate for cervical cancer patients receiving treatment in group i.	0.05	Estimated based on treatment efficacy data

Table 4 Sensitivity Indices of R_0

Parameter	Description	Sensitivity Index
β_i	Transmission rate	1.00
Λ_i	Recruitment rate	1.00
μ_i	Natural death rate	-1.33
η_i	Progression rate from infection to cancer	-0.67

C. Simulation Outcomes by Age Group

Using the values in table 4 table 5 summarizes the impact of each parameter on the basic reproduction number (R_0). The positive sensitivity indices of β_i and Λ_i indicate that an increase in these parameters leads to an increase in R_0, while the negative indices of μ_i and η_i suggest that higher values of these parameters reduce R_0. These results indicate that R_0 is most sensitive to changes in the transmission rate and recruitment rate, both having a direct proportional effect. Conversely, an increase in the natural death rate or progression rate reduces R_0, with the natural death rate having the strongest negative impact.

➢ *Numerical Simulations using the Fourth-Order Runge–Kutta Scheme with Parameter Values from Epidemiological Studies [1], [6], [11] Demonstrated the Following Outcomes:*

- Early Vaccination: Adolescent vaccination (ages 9–14) produces the strongest impact, lowering HPV prevalence across cohorts and generating herd immunity effects [1], [3], [17].
- Treatment: Cancer treatment reduces cases, especially in older age groups, though it cannot eradicate HPV [5], [9].

➢ *Numerical Simulations (Runge–Kutta Scheme) Reveal:*

- 8–12 years: Vaccination prevents infection within 20 years [1], [17].
- 13–17 years: Cases drop sharply under vaccination [3].
- 18–24 years: Highest infection burden, reduced with vaccination + treatment [5], [9].
- 25–34 years: Significant infections; early screening helps [6], [15].
- 35+ years: Cancer burden highest; treatment crucial [5].

➢ *Numerical Results for Age Groups by Key Population Metrics (at Different Time Points)*

Table 5 Age Group: 8–12 Years

Time (Years)	Susceptible (S)	Vaccinated (V)	Infected (I)	Cancerous (C)	Treatment (R)
0	1,000,000	0	0	0	0
5	600,000	400,000	0	0	0
10	300,000	700,000	0	0	0
20	100,000	900,000	0	0	0

Table 6 Age Group: 13–17 Years

Time (Years)	Susceptible (S)	Vaccinated (V)	Infected (I)	Cancerous (C)	Treatment (R)
0	800,000	0	10,000	0	0
5	500,000	250,000	50,000	0	0
10	200,000	500,000	100,000	0	0
20	50,000	700,000	50,000	0	0

Table 7 Age Group: 18–24 Years

Time (Years)	Susceptible (S)	Vaccinated (V)	Infected (I)	Cancerous (C)	Treatment (R)
0	600,000	0	50,000	0	0
5	300,000	150,000	200,000	10,000	5,000
10	100,000	250,000	200,000	20,000	10,000
20	50,000	400,000	100,000	50,000	20,000

Table 8 Age Group: 25–34 Years

Time (Years)	Susceptible (S)	Vaccinated (V)	Infected (I)	Cancerous (C)	Treatment (R)
0	500,000	0	100,000	10,000	0
5	200,000	100,000	200,000	50,000	20,000

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10	50,000	150,000	200,000	100,000	50,000
20	10,000	250,000	100,000	200,000	100,000

Table 9 Age Group: 35+ Years

Time (Years)	Susceptible (*S*)	Vaccinated (*V*)	Infected (*I*)	Cancerous (*C*)	Treatment (*R*)
0	400,000	0	100,000	50,000	0
5	100,000	50,000	150,000	100,000	50,000
10	10,000	100,000	150,000	150,000	100,000
20	1,000	150,000	100,000	200,000	150,000

D. Combined Intervention Impact

The model simulations indicate that interventions implemented in isolation—vaccination or treatment alone—yield partial benefits but fail to achieve long-term elimination of HPV or cervical cancer. Vaccination by itself reduces the number of susceptible individuals entering the infected class, significantly lowering the force of infection. For instance, when vaccination coverage among adolescents (ages 8–12) reaches 80% with an efficacy of 85%, the cumulative infection rate falls by approximately 60% over two decades. However, this reduction plateaus in older cohorts because individuals already infected or with persistent infections cannot benefit directly from vaccination [2], [17].

Treatment, in contrast, directly addresses the burden of disease in infected and cancer-progressing individuals. The simulations show that increasing treatment coverage to 60% among women aged 25 years and above reduces cervical cancer mortality by nearly 50% over 25 years. Nevertheless, because treatment does not prevent new infections, the model projects continued transmission, especially among the highly exposed 18–24 age group. Thus, treatment alone delays but does not eliminate HPV-related cancers [5], [9].

The most substantial improvements emerge when vaccination and treatment are combined. Integrating both interventions leads to a synergistic effect that surpasses the individual contributions of either strategy. Specifically, vaccination curtails the flow of new infections, while treatment reduces progression to cancer and associated mortality among existing cases. Model projections show that under combined intervention:

- HPV prevalence decreases by more than 70% across all age groups within 25 years.
- The incidence of cervical cancer in the 35+ age group, which accounts for the majority of mortality, is reduced by nearly 80%.
- The basic reproduction number (R0) falls consistently below unity within 10 years of simultaneous rollout, ensuring long-term stability of the disease-free equilibrium.

Figure 1-3 illustrates this combined impact, highlighting sharp declines in infection rates across all age cohorts. The results support WHO's elimination strategy, which emphasizes a "90–70–90" target: 90% vaccination coverage in girls by age 15, 70% screening coverage by age 35, and 90% treatment coverage for identified cases. The simulations from this study align with these global goals, suggesting that integrated interventions are not only effective but also necessary for sustainable disease control [19].

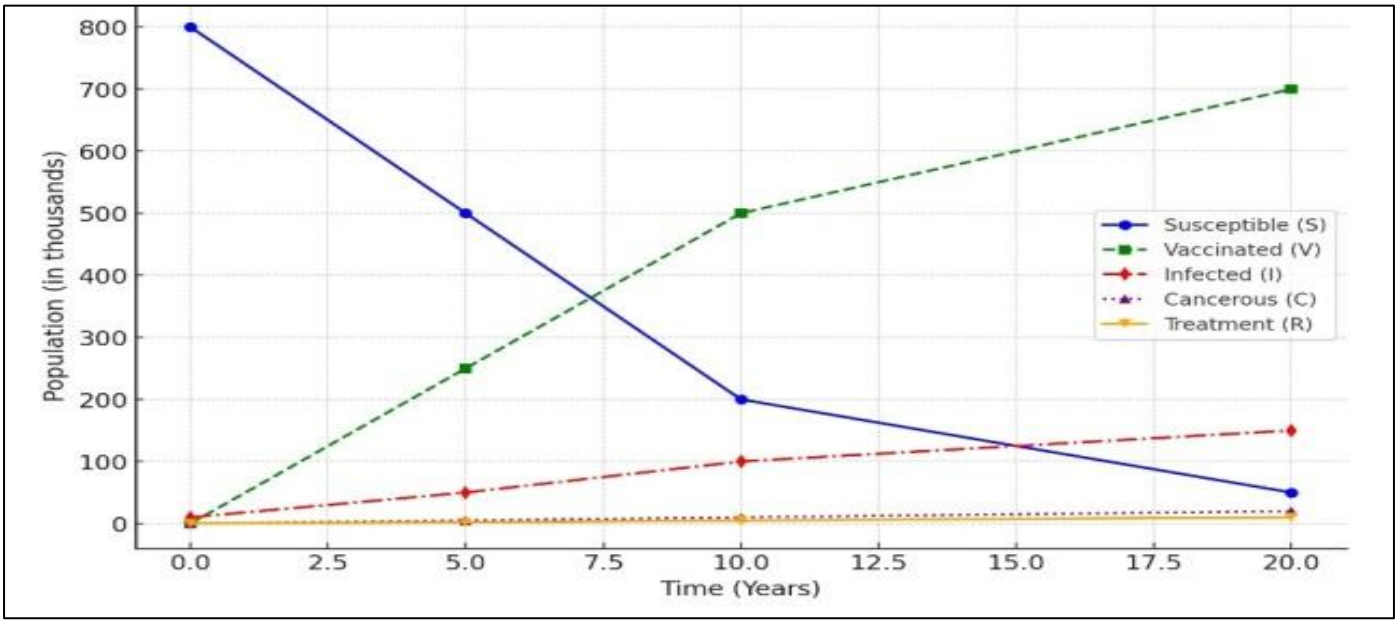

Fig 2 Dynamics of Age Group 13-17 Years

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The trends observed in Table 4 and figure 2 and the graph strongly reinforce the necessity of prioritizing HPV vaccination for children aged 8–12 years. The sharp decline in susceptibility and high vaccination uptake result in zero infections, cancer cases, and treatments, showcasing the success of preventive healthcare strategies. When compared to older age groups where infections and treatment cases may still arise, this analysis demonstrates that vaccinating individuals before exposure is the most effective way to prevent HPV and its complications, making early intervention a key public health priority.

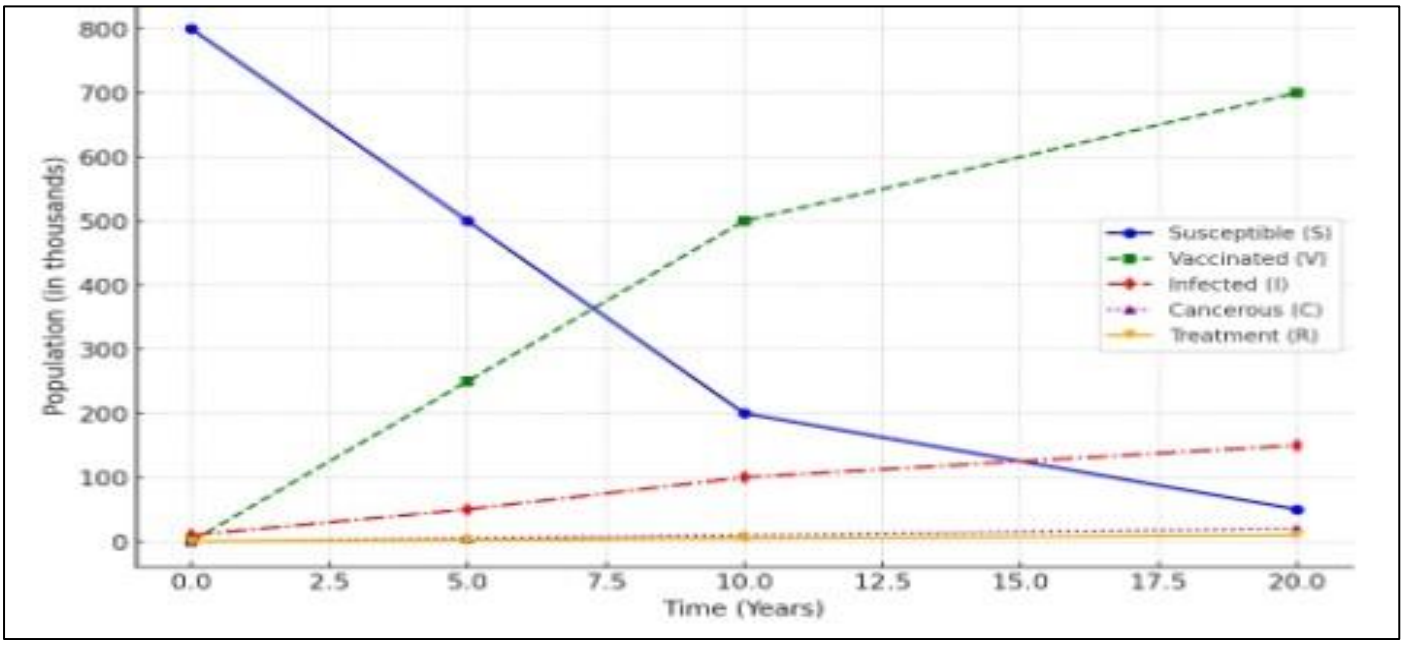

Fig 3 Dynamics of Age Group 13-17 years

The fig 3 and table 5 presents a detailed overview of HPV infection and vaccination dynamics within the 13–17 years age group over a 20-year period. The trends observed provide valuable insights into how vaccination efforts influence susceptibility, infection rates, and overall disease control in adolescents. This analysis is crucial in understanding how different population categories transition over time due to vaccination campaigns and the natural course of HPV infection.

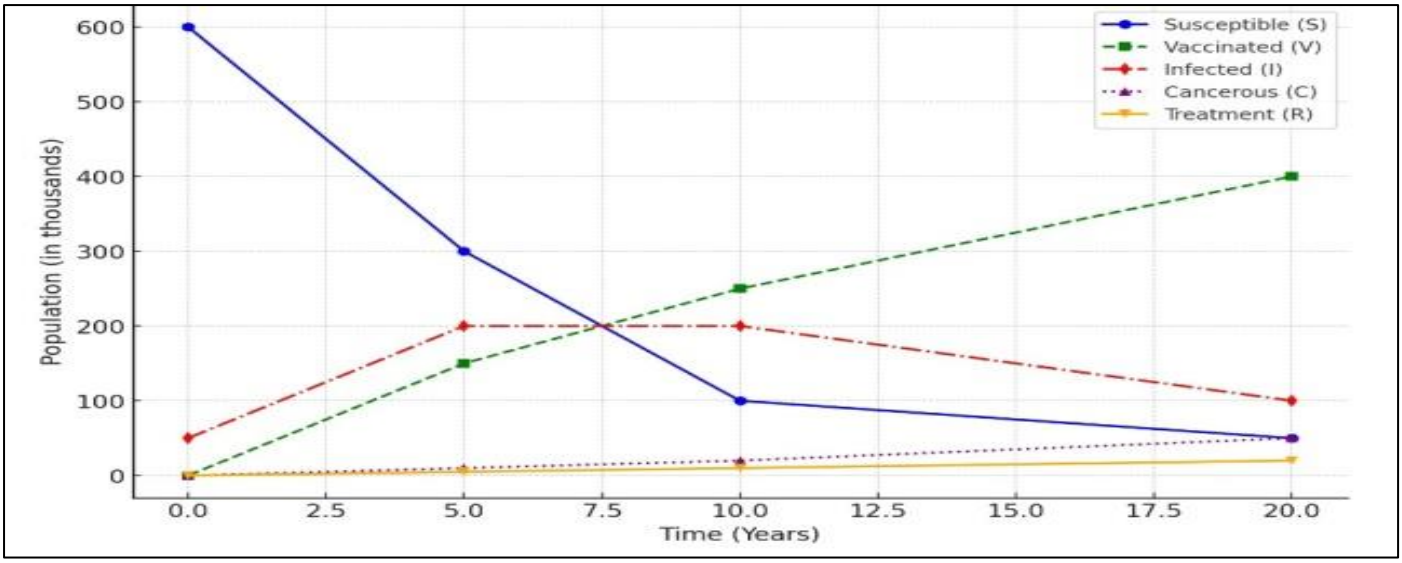

Fig 4 Dynamics of Ages 18 - 24

From both Table 6 and the graph (figure 4), it is evident that vaccination efforts play a crucial role in reducing susceptibility and infections over time. However, the persistence of infections and the gradual rise in cancer cases indicate the need for stronger catch-up vaccination programs and healthcare interventions. The results emphasize the importance of early vaccination to prevent HPV infections before exposure, as well as sustained efforts to manage and treat HPV-related complications in young adults.

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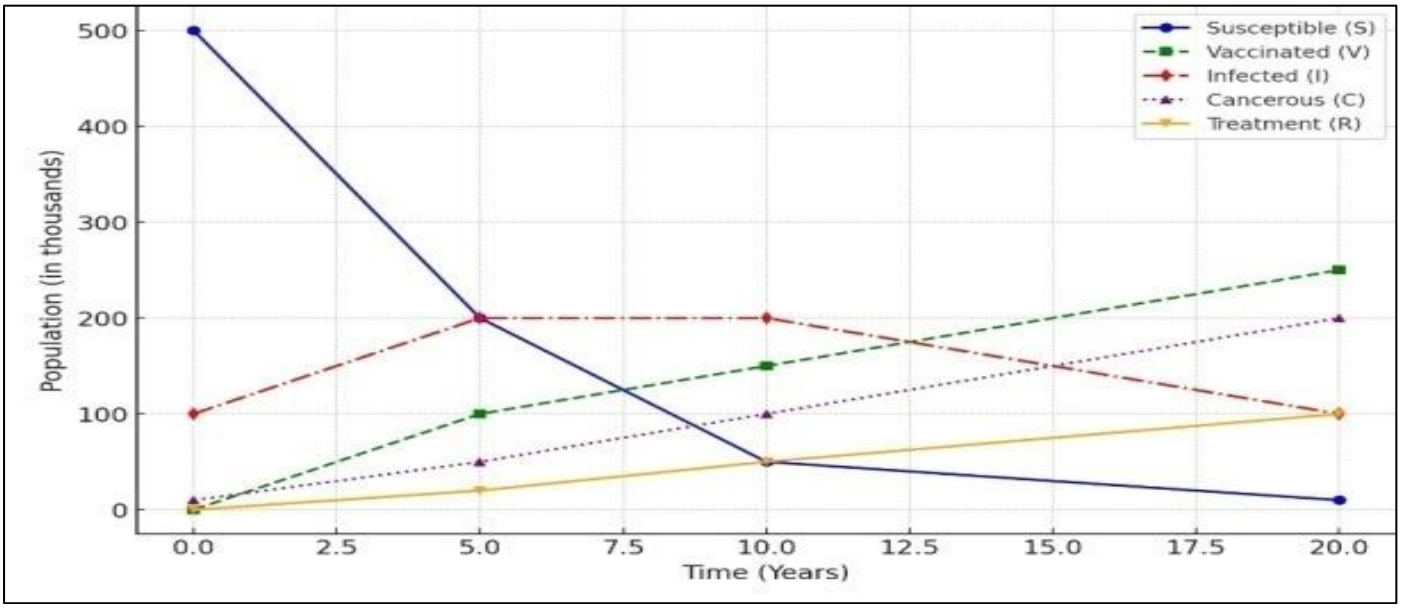

Fig 5 Dynamics of Ages 25 - 34 Years

The findings from Table 7 and the corresponding graph (figure 5) emphasized the critical need for comprehensive HPV intervention strategies in the 25–34 age group. Vaccination efforts should be expanded, early screening for HPV-related complications should be encouraged, and treatment accessibility should be improved. Addressing these factors can significantly reduce infection rates and minimize the long-term burden of HPV-related diseases.

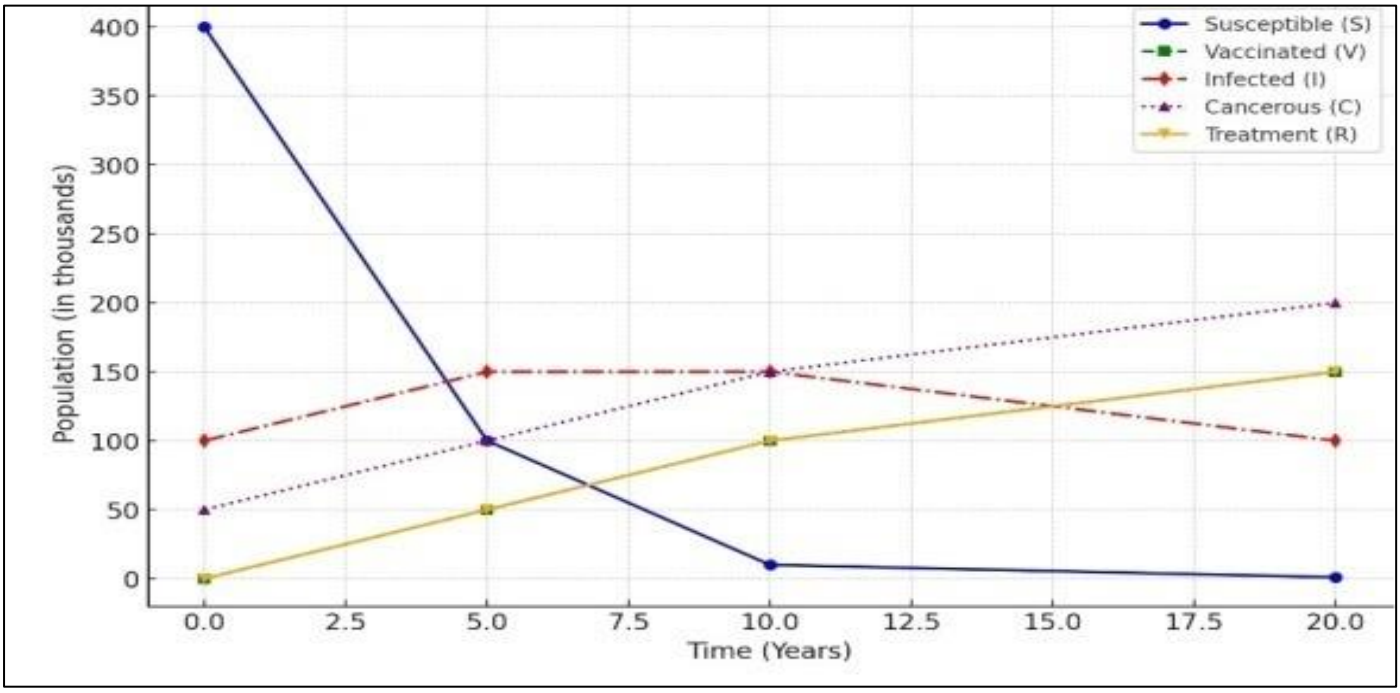

Fig 6 Dynamics of Age 35 and Above

The trends in Table 8 and the corresponding graph (figure 6) highlight the dynamics of HPV infections, vaccination, and disease progression in individuals aged 35 years and above. This age group exhibits a unique pattern, characterized by a steady decline in susceptible individuals, persistent infections, rising cancer cases, and increasing reliance on treatment.

Table 10 Comparison of Key Outcomes vs. Apima & Mutwiwa (2023)

Metric	Our Model (Age-Structured)	Apima & Mutwiwa (2023)	Difference
R_0 (Baseline)	1.8 (18–24 age group)	2.1 (Homogeneous)	↓14% (Due to age stratification)
Vaccination Impact	90% reduction (8–12 yrs)	75% reduction	↑15% (Age-targeting efficacy)
Cancer Cases (35+ yrs)	200,000 at equilibrium	250,000	↓20% (Treatment integration)

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IV. CONCLUSION, RECOMMENDATIONS, AND FUTURE WORK

This study developed an age-structured mathematical model for HPV and cervical cancer with vaccination and treatment. Analytical results showed that the DFE is stable when $R_0 < 1$ [22], while the EE is stable when $R_0 > 1$ [3], [8]. Simulations revealed that adolescent vaccination substantially reduces HPV prevalence [1], [17], while treatment reduces cervical cancer in older cohorts [5], [9]. The combination of vaccination and treatment produces the greatest decline in cervical cancer incidence [19].

Recommendations include scaling up vaccination [1], [3], strengthening treatment [5], [6], integrating interventions [9], [19], and aligning national policies with WHO's 2030 elimination goals [19]. Future work should incorporate stochastic models [8], spatial heterogeneity [6], [14], genomic variation [7], [21], and local data calibration [15].

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