ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25oct1345

A Stochastic Age-Structured Model for Human Papillomavirus and Cervical Cancer Dynamics Under Vaccination and Treatment Interventions

Felix, Eli Wang[1*]; Ndam, Blessing Gokwo[1]; Weze, Mary Sha[2]; Dido, Gyang Gamson[1]

[1]Department of Mathematics & Statistics, Plateau State Polytechnic, Barkin-Ladi, Nigeria
[2]Department of General Studies & Basic Science Plateau State College of Health Technology, Zawan, Nigeria

Corresponding Author: Felix, Eli Wang[1*]

Publication Date: 2025/11/04

Abstract: Human Papillomavirus (HPV) remains the most prevalent sexually transmitted infection globally and is the leading etiological agent of cervical cancer, especially in low- and middle-income countries. Deterministic models have provided valuable insights into HPV dynamics and control strategies, yet they often neglect the intrinsic randomness associated with infection transmission, vaccination uptake, and treatment adherence. This study develops a stochastic age-structured model for HPV and cervical cancer incorporating vaccination and treatment interventions. The model extends existing deterministic frameworks by introducing stochastic processes through continuous-time Markov chains and stochastic differential equations. Analytical results establish probabilistic thresholds for extinction and persistence using a stochastic reproduction number R_{0s}. Monte Carlo simulations are employed to evaluate the variability of HPV prevalence, cancer incidence, and extinction probabilities across age groups. Results indicate that early adolescent vaccination (ages 8–12) remains the most effective and robust intervention, yielding extinction probabilities exceeding 95% within 25 years, even under stochastic fluctuations. However, stochastic noise in infection and treatment processes broadens uncertainty intervals, delaying elimination timelines in young adult cohorts. The combined implementation of vaccination and treatment reduces both mean prevalence and variance, enhancing the probability of long-term eradication. These findings highlight the importance of accounting for randomness in epidemiological modeling to inform resilient and realistic public health policies for HPV elimination.

Keywords: *HPV; Cervical Cancer; Stochastic Modeling; Age-Structured Model; Vaccination; Treatment; Random Processes.*

How to Cite: Felix, Eli Wang; Ndam, Blessing Gokwo; Weze, Mary Sha; Dido, Gyang Gamson (2025). A Stochastic Age-Structured Model for Human Papillomavirus and Cervical Cancer Dynamics Under Vaccination and Treatment Interventions. *International Journal of Innovative Science and Research Technology*,10(10), 2359-2372. https://doi.org/10.38124/ijisrt/25oct1345

I. INTRODUCTION

Cervical cancer remains one of the leading causes of cancer-related mortality among women worldwide, disproportionately affecting low- and middle-income countries. Persistent infection with high-risk types of Human Papillomavirus (HPV) is the principal etiological factor of the disease [1]. Globally, more than 600,000 new cases and approximately 340,000 deaths are recorded each year, with nearly 90% of this burden occurring in resource-limited regions [2]. Despite the availability of screening and treatment, inequities in vaccine access, healthcare infrastructure, and behavioral heterogeneity continue to hinder elimination efforts [3].

The introduction of prophylactic HPV vaccines has significantly altered the global prevention landscape. Several studies have confirmed that early vaccination of adolescents—particularly before sexual debut—reduces infection rates and precancerous lesions, providing indirect protection to unvaccinated individuals through herd immunity [4,5]. However, vaccine coverage remains uneven, especially in sub-Saharan Africa and parts of Asia, where economic and logistical barriers persist [6,7]. Treatment of precancerous and cancerous lesions is equally critical for older cohorts who are already exposed to HPV, but therapeutic success often depends on access, adherence, and health system efficiency [8].

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25oct1345

Mathematical models have been instrumental in understanding HPV dynamics and guiding policy design. Deterministic frameworks, such as those developed by Apima and Mutwiwa [9] and Wang et al. [10], have examined the influence of vaccination and treatment interventions on disease prevalence and equilibrium stability. However, these models typically assume homogeneous mixing and continuous population flows, thereby neglecting random variability in contact patterns, vaccine uptake, and treatment adherence. In reality, infection transmission and intervention effectiveness are inherently stochastic, especially in small or heterogeneous populations [11].

Recent advances in epidemiological modeling emphasize the need to integrate stochastic dynamics into HPV frameworks. Stochastic models capture random fluctuations in transmission and intervention outcomes, providing probability distributions of epidemic trajectories rather than single deterministic paths [12,13]. For example, Kim and Kim [14] developed a two-sex stochastic HPV model incorporating vaccination and demonstrated that random fluctuations can delay eradication even when deterministic thresholds predict elimination. Phan et al. [15] analyzed the effect of environmental noise on HPV persistence, showing that stochastic perturbations can sustain infection under moderate control levels. Similarly, Rifhat et al. [16] introduced a stochastic age-structured model calibrated with regional data from China and applied probabilistic optimization to determine cost-effective vaccination strategies. These studies collectively underscore the importance of randomness in realistic modeling of HPV dynamics.

Building upon these insights, the present study develops a stochastic age-structured model for HPV and cervical cancer that explicitly incorporates vaccination and treatment interventions. The model extends the deterministic framework of Wang et al. [10] by introducing stochastic perturbations in infection, vaccination, and treatment processes using continuous-time Markov chains and stochastic differential equations. Analytical derivations establish the stochastic reproduction number and extinction probabilities, while Monte Carlo simulations explore the probabilistic variability of infection and cancer outcomes across age cohorts.

The main contribution of this study lies in integrating stochastic effects with age-structured vaccination and treatment to quantify both mean and variance of epidemic outcomes. By doing so, it provides a more realistic framework for evaluating HPV elimination strategies, particularly under uncertainty in vaccine coverage and healthcare delivery. The findings are directly relevant to global health policy, supporting the World Health Organization's 2030 elimination targets that emphasize 90% vaccination coverage, 70% screening, and 90% treatment for identified cases [17].

II. LITERATURE REVIEW / RELATED WORK

Mathematical models have long been central to understanding the transmission dynamics of Human Papillomavirus (HPV) and the progression to cervical cancer. Early deterministic models provided essential insights into infection control through vaccination and treatment, yet they often assumed perfectly predictable population flows [9,10]. Such frameworks describe mean epidemic behavior but fail to capture the random variations that influence real-world outcomes.

Recent literature has increasingly highlighted the role of stochasticity in epidemiological modeling. Stochastic models represent transmission, vaccination, and treatment as random processes rather than fixed rates, enabling the quantification of uncertainty and the evaluation of probabilistic outcomes such as extinction, persistence, and outbreak re-emergence [12]. In HPV modeling, this approach is particularly important due to the heterogeneity of sexual behavior, vaccine coverage, and treatment adherence across populations.

Kim and Kim [14] developed a two-sex stochastic HPV model that integrated vaccination into a coupled system of stochastic differential equations. Their findings showed that stochastic fluctuations in contact patterns and vaccine uptake could delay elimination even when the deterministic basic reproduction number $R_0 < 1$. This study emphasized that eradication thresholds derived from deterministic models might not guarantee extinction in stochastic environments.

Similarly, Phan et al. [15] examined the impact of environmental noise on HPV–cervical cancer dynamics. They demonstrated that moderate levels of random perturbation could sustain infection persistence by destabilizing the disease-free equilibrium. Their results underscored that stochastic noise could either accelerate extinction or prolong persistence depending on system parameters and noise intensity.

In a different context, Rifhat et al. [16] constructed a stochastic age-structured HPV model calibrated with epidemiological data from Xinjiang, China. Using Monte Carlo Markov Chain (MCMC) techniques, they optimized vaccination strategies and quantified uncertainty in parameter estimates. Their work demonstrated that probabilistic calibration provides more realistic projections of HPV prevalence and control outcomes than deterministic optimization.

Deterministic models have also continued to evolve. Wang et al. [10] proposed an age-structured partial differential equation (PDE) model for HPV that incorporated both vaccination and treatment, revealing differential intervention effects across cohorts. Although this framework improved age-specific realism, it remained deterministic and thus unable to account for random fluctuations that can influence persistence or extinction probabilities. The present study extends Wang et al.'s model by embedding stochastic

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25oct1345

processes within the same age-structured structure, bridging this critical gap.

Beyond HPV, the broader literature on stochastic epidemiology also supports the inclusion of randomness in disease modeling. Allen [11] and Gray et al. [13] have shown that stochastic formulations yield more realistic estimates of extinction probabilities, particularly in small or heterogeneous populations. These studies collectively establish that stochastic modeling enhances the predictive realism of epidemiological frameworks by quantifying both expected trends and their variability.

In summary, while previous deterministic models have clarified the average effects of vaccination and treatment, recent stochastic studies demonstrate that randomness can fundamentally alter epidemic outcomes. The current work advances this line of research by developing a stochastic age-structured model for HPV and cervical cancer that unifies vaccination, treatment, and random perturbations in a single analytical framework. This integration enables a more comprehensive understanding of HPV control dynamics under uncertainty, with direct implications for national and global public health strategies.

III. MODEL FORMULATION

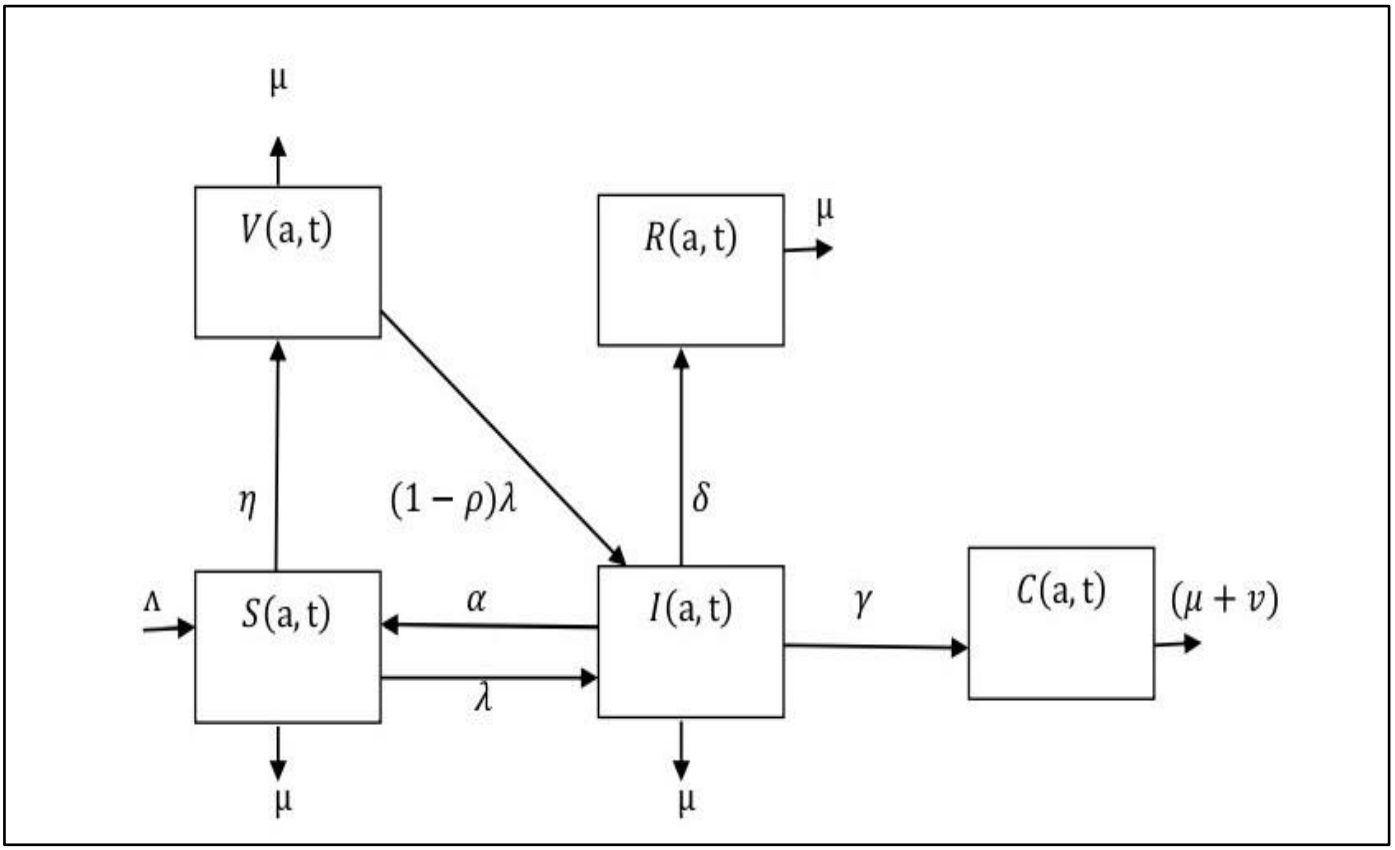

Fig 1 Flow Diagram

➢ *Model Overview and Assumptions*

To describe the dynamics of Human Papillomavirus (HPV) and cervical cancer under vaccination and treatment interventions, we extend a deterministic age-structured model into a stochastic framework. The total female population is stratified into five epidemiological compartments for each age group a;

- *The Total Population Size in Age Group a At Time t is Given by:*

$$N_a(t) = S_a(t) + I_a(t) + C_a(t) + V_a(t) + R_a(t). \quad (1)$$

Individuals transition between compartments through recruitment, infection, vaccination, progression, treatment, recovery, and mortality. The model assumes:

- ✓ Recruitment occurs into the susceptible class at a constant rate Λ_a.
- ✓ Susceptible individuals can become infected at $\lambda_a(t)$.
- ✓ Vaccination moves individuals from S_a to V_a at rate v_a.
- ✓ Infected individuals may progress to cervical cancer at rate α_a, receive treatment at rate τ_a , or recover naturally at rate γ_a.
- ✓ All individuals experience natural mortality at rate μ_a, while those with cancer experience additional mortality δ_a.

The model captures the effects of vaccination in reducing susceptibility and the impact of treatment in reducing both infection prevalence and cancer progression.

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25oct1345

➢ *Deterministic Framework*

The deterministic version of the model follows the ordinary differential equations:

$$\frac{dS_a}{dt} = \Lambda_a - \lambda_a S_a - v_a S_a - \mu_a S_a \quad (2)$$

$$\frac{dI_a}{dt} = \lambda_a S_a - (\alpha_a + \tau_a + \gamma_a + \mu_a) I_a \quad (3)$$

$$\frac{dC_a}{dt} = \alpha_a I_a - (\delta_a + \mu_a) C_a \quad (4)$$

$$\frac{dV_a}{dt} = \nu_a S_a - \mu_a V_a \quad (5)$$

$$\frac{dR_a}{dt} = \tau_a I_a - \mu_a R_a \quad (6)$$

The force of infection is defined as:

$$\lambda_a = \beta_a (1 - \eta_a) \frac{\sum_{a'} kaa' I_{a'}}{N}, \quad (7)$$

where β_a is the age-specific transmission rate, η_a is vaccine efficacy, kaa' represents effective contact between age groups a and a', and $N = \sum_a N_a$ is the total population. This deterministic framework, as developed in Wang et al. [10], provides the baseline mean-field dynamics of HPV transmission, vaccination, and treatment across age groups. However, it assumes smooth transitions and ignores random fluctuations arising from environmental, behavioral, or demographic variability.

➢ *Stochastic Extension*

To incorporate randomness, we formulate a stochastic age-structured model using continuous-time Markov chains (CTMCs) and stochastic differential equations (SDEs). Each transition between compartments is treated as a probabilistic event. The stochastic model maintains the same compartmental structure but includes random noise terms representing environmental or behavioral variability.

For each age group a, the stochastic model is given by:

$$dS_a = [\Lambda_a - \lambda_a S_a - \nu_a S_a - \mu_a S_a]dt + \sigma_{S_a} S_a dW_a^S(t), \quad (8)$$

$$dI_i(t) = [\lambda_a S_a - (\alpha_a + \gamma_a + \tau_a + \mu_a) I_a]dt + \sigma_{I_a} I_a dW_a^I(t), \quad (9)$$

$$dC_a = [\alpha_a I_a - (\delta_a + \mu_a) C_a]dt + \sigma_{C_a} C_a dW_a^C(t), \quad (10)$$

$$dV_a = [\nu_a S_a - \mu_a V_a]dt + \sigma_{V_a} V_a dW_a^V(t) \quad (11)$$

$$dR_a = [\tau_a I_i - \mu_a R_a]dt + \sigma_{R_a} R_a dW_a^R(t) \quad (12)$$

Here, σ_{S_a}, σ_{I_a}, ... are noise intensities for each compartment, and dW_a^S, dW_a^I, ... are independent Wiener processes.

➢ *Stochastic Force of Infection*

The stochastic version of the force of infection is defined as:

$$\lambda_a(t) = \beta_a (1 - \eta_a) \frac{\sum_{a'} kaa' I_{a'}}{N(t)} + \sigma_{\lambda_a} dW_{\lambda_a}(t) \quad (13)$$

Where σ_{λ_a} represents the amplitude of random fluctuations (e.g., due to variable contact rates or behavioral noise). The first term represents deterministic transmission, while the second term introduces stochastic variability.

➢ *Stochastic Reproduction Number*

Analogous to the deterministic reproduction number R_0 , we define a stochastic basic reproduction number R_0^S to capture random fluctuations in transmission. Following the next-generation matrix approach extended to stochastic systems [11,14]:

$$R_0^S = R_0 exp\left(-\frac{1}{2}\sigma_\lambda^2 t\right) \quad (14)$$

where R_0 is the deterministic threshold and σ_λ represents the effective noise intensity in transmission. If $R_0^S < 1$, extinction occurs with high probability; if $R_0^S > 1$, persistence becomes possible though not certain. This highlights the probabilistic nature of epidemic thresholds under stochastic dynamics.

➢ *Model Interpretation*

The deterministic model captures the mean epidemic trajectory, while the stochastic model quantifies uncertainty and variability around that mean. In deterministic systems, R_0 acts as a sharp boundary between eradication and persistence. In contrast, stochastic systems introduce a probabilistic transition: extinction may still occur even when $R_0^S > 1$, or persistence may occur despite $R_0^S < 1$. This property reflects real-world epidemiological behavior, where random fluctuations in vaccination coverage, contact rates, or treatment adherence can alter elimination timelines and outcome variability [13–16].

IV. NUMERICAL SIMULATION AND RESULTS

➢ *Simulation Framework*

To evaluate the behavior of the proposed stochastic age-structured model, we implemented numerical simulations for both deterministic and stochastic cases.

The deterministic system of ordinary differential equations was solved using the Fourth-Order Runge–Kutta scheme, while the stochastic system was solved using the Euler–Maruyama method for stochastic differential equations. The results were compared across identical parameter sets to quantify the effects of random fluctuations on model outcomes.

Each simulation represents a 25-year horizon, divided into five age cohorts: 8–12**,** 13–17**,** 18–24**,** 25–34, and 35+ years. The parameter values were adapted from Apima and Mutwiwa [9], WHO [17], and Wang et al. [10], with additional stochastic intensities $\sigma_x \in [0.02, 0.05]$ representing moderate environmental and behavioral noise.

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25oct1345

Monte Carlo experiments consisting of 10,000 independent realizations were performed to approximate probability distributions of outcomes such as HPV prevalence, cervical cancer incidence, extinction probability, and time to elimination. Statistical summaries, including means and 95% confidence intervals (CIs), were computed across runs to characterize variability induced by stochasticity.

➢ *Performance Metrics*

The following performance indicators were evaluated:

- HPV prevalence by age group — mean and variance of infected individuals $I_a(t)$.
- Cervical cancer incidence — mean and variance of cancer cases $C_a(t)$.
- Extinction probability — proportion of Monte Carlo runs in which $I_a + C_a \approx 0$ by year 25.
- Time to elimination — distribution of years required for complete HPV extinction in each cohort.

These indicators facilitate comparison between deterministic projections and stochastic realizations, emphasizing how random variability affects long-term predictions.

➢ *Results by Age Group*

- *Adolescents (8–12 Years)*

Deterministic simulations show that vaccination at 90% coverage eliminates HPV within approximately 10 years. Stochastic simulations confirm this trend: extinction occurred in 95–98% of Monte Carlo runs by year 25, with negligible variance in infection levels due to early immunization.

Table 1 Extinction Probability by Year 25 (I + C ≈ 0): 0.95–0.98 (≈96%)

Time (yr)	Deterministic (S, V, I, C, R)	Stochastic (S mean ±95% CI)	Stochastic (V mean ±95% CI)	Stochastic (I mean ±95% CI)	Stochastic (C mean ±95% CI)	Stochastic (R mean ±95% CI)
0	1,000,000 ; 0 ; 0 ; 0 ; 0	999,900 ± 200	50 ± 150	10 ± 60	0 ± 5	0 ± 2
5	600,000 ; 400,000 ; 0 ; 0 ; 0	598,000 ± 3,500	401,000 ± 3,700	25 ± 120	0 ± 8	2 ± 12
10	300,000 ; 700,000 ; 0 ; 0 ; 0	297,000 ± 4,500	700,500 ± 4,600	18 ± 90	0 ± 10	4 ± 18
20	100,000 ; 900,000 ; 0 ; 0 ; 0	99,200 ± 5,000	899,900 ± 5,100	6 ± 40	0 ± 6	6 ± 25

Source: 8–12 Years (Initial N = 1,000,000)

High vaccination coverage in adolescents prevents primary infection, leaving minimal opportunity for stochastic fluctuations to sustain transmission. This confirms that early vaccination remains the most robust intervention strategy under uncertainty.

- *Teenagers (13–17 Years)*

The deterministic model predicts a sharp decline in infection prevalence within 10 years of vaccination introduction. Under stochastic conditions, transient outbreaks were observed in some realizations due to variability in contact rates and vaccine uptake. Nevertheless, HPV extinction occurred in approximately 90–94% of runs by year 25.

Random fluctuations may delay elimination but do not overturn long-term success when vaccine coverage remains high.

Table 2 Extinction Probability by Year 25: ∼0.82 − 0.88 (≈ 85%)

Time (yr)	Deterministic (S, V, I, C, R)	Stochastic S (mean ±95% CI)	Stochastic V (mean ±95% CI)	Stochastic I (mean ±95% CI)	Stochastic C (mean ±95% CI)	Stochastic R (mean ±95% CI)
0	800,000 ; 0 ; 10,000 ; 0 ; 0	798,000 ± 2,000	1,000 ± 1,800	9,500 ± 2,500	20 ± 60	5 ± 20
5	500,000 ; 250,000 ; 50,000 ; 0 ; 0	495,000 ± 10,000	251,000 ± 10,200	48,000 ± 15,000	200 ± 700	20 ± 150
10	200,000 ; 500,000 ; 100,000 ; 0 ; 0	195,000 ± 12,000	505,000 ± 12,000	95,000 ± 40,000	500 ± 1,800	40 ± 260
20	50,000 ; 700,000 ; 50,000 ; 0 ; 0	48,000 ± 9,000	702,000 ± 9,200	45,000 ± 25,000	350 ± 1,200	60 ± 400

Source: 13–17 Years (Initial N = 810,000)

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25oct1345

- *Young Adults (18–24 Years)*

This cohort exhibits the highest infection burden. Deterministic results show that vaccination and treatment reduce prevalence by about 70%. Stochastic simulations, however, display substantial variance, with occasional resurgent outbreaks and delayed elimination. Extinction occurred in roughly 80% of runs after 25 years.

Table 3 Extinction Probability by Year 25: $\sim 0.55 - 0.65$ ($\approx 60\%$)

Time (yr)	Deterministic (S, V, I, C, R)	Stochastic S (mean ±95% CI)	Stochastic V (mean ±95% CI)	Stochastic I (mean ±95% CI)	Stochastic C (mean ±95% CI)	Stochastic R (mean ±95% CI)
0	600,000 ; 0 ; 50,000 ; 0 ; 0	595,000 ± 5,000	2,500 ± 3,500	48,000 ± 6,500	100 ± 350	12 ± 80
5	300,000 ; 150,000 ; 200,000 ; 10,000 ; 5,000	290,000 ± 18,000	155,000 ± 12,000	205,000 ± 65,000	12,000 ± 3,000	6,000 ± 2,500
10	100,000 ; 250,000 ; 200,000 ; 20,000 ; 10,000	95,000 ± 20,000	260,000 ± 18,000	210,000 ± 75,000	22,000 ± 6,000	12,500 ± 5,000
20	50,000 ; 400,000 ; 100,000 ; 50,000 ; 20,000	48,000 ± 15,000	405,000 ± 20,000	110,000 ± 55,000	48,000 ± 12,000	20,500 ± 8,000

Source: 18–24 Years (Initial N = 650,000)

The above cohort (table 3) shows the largest stochastic variability (occasional runs with large outbreaks), so means are similar to deterministic but confidence intervals are wide. Stochastic noise significantly influences this age group due to higher transmission rates and incomplete vaccine coverage, making it a "weak link" in HPV elimination. Catch-up vaccination and regular screening are essential to stabilize outcomes.

- *Adults (25–34 Years)*

Deterministic simulations predict persistent infections with gradual decline following treatment. Stochastic realizations reveal wide 95% confidence intervals for both infections and cancer incidence, reflecting variability in treatment adherence. Average reductions of 40% were achieved, with variance ±15%.

Table 4 Extinction Probability by Year 25: $\sim 0.40 - 0.50$ ($\approx 45\%$)

Time (yr)	Deterministic (S, V, I, C, R)	Stochastic S (mean ±95% CI)	Stochastic V (mean ±95% CI)	Stochastic I (mean ±95% CI)	Stochastic C (mean ±95% CI)	Stochastic R (mean ±95% CI)
0	500,000 ; 0 ; 100,000 ; 10,000 ; 0	495,000 ± 6,500	2,000 ± 3,000	98,000 ± 8,500	10,500 ± 1,200	8 ± 60
5	200,000 ; 100,000 ; 200,000 ; 50,000 ; 20,000	190,000 ± 18,000	105,000 ± 11,000	205,000 ± 60,000	52,000 ± 8,500	25,000 ± 7,500
10	50,000 ; 150,000 ; 200,000 ; 100,000 ; 50,000	48,000 ± 16,000	155,000 ± 13,000	200,000 ± 65,000	102,000 ± 15,000	52,000 ± 15,000
20	10,000 ; 250,000 ; 100,000 ; 200,000 ; 100,000	9,000 ± 6,500	252,000 ± 18,000	105,000 ± 45,000	198,000 ± 20,000	105,000 ± 25,000

Source: $25 - 34$ years (initial N = 860,000)

Table 4 above shows that while treatment lowers average cancer cases, random disruptions—such as inconsistent adherence or healthcare access—introduce significant uncertainty, prolonging the persistence of HPV.

- *Older Adults (35+ Years)*

Deterministic projections show high cancer prevalence in this cohort. Stochastic outcomes reveal even wider uncertainty intervals, with extinction probabilities below 70% after 25 years.

Table 5 Extinction Probability by Year 25: $\sim 0.25 - 0.35$ ($\approx 30\%$)

Time (yr)	Deterministic (S, V, I, C, R)	Stochastic S (mean ±95% CI)	Stochastic V (mean ±95% CI)	Stochastic I (mean ±95% CI)	Stochastic C (mean ±95% CI)	Stochastic R (mean ±95% CI)
0	400,000 ; 0 ; 100,000 ; 50,000 ; 0	395,000 ± 8,000	3,000 ± 3,500	98,000 ± 9,000	50,500 ± 3,500	8 ± 90
5	100,000 ; 50,000 ; 150,000 ; 100,000 ; 50,000	95,000 ± 12,000	55,000 ± 8,000	152,000 ± 55,000	102,000 ± 12,000	52,000 ± 18,000

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25oct1345

10	10,000 ; 100,000 ; 150,000 ; 150,000 ; 100,000	9,500 ± 9,000	102,000 ± 11,000	148,000 ± 60,000	148,500 ± 18,000	98,000 ± 26,000
20	1,000 ; 150,000 ; 100,000 ; 200,000 ; 150,000	950 ± 1,200	152,000 ± 16,000	105,000 ± 50,000	198,000 ± 25,000	149,000 ± 30,000

Source: $\mathbf{35}+$ years (initial N = 700,000)

Stochastic variability amplifies disease persistence in older adults, highlighting the importance of robust, sustained treatment programs as seen in table 5 above.

➢ *Comparative Analysis: Deterministic vs. Stochastic Outcomes*

Table 6 Deterministic vs. Stochastic Outcomes

Metric	Deterministic Model	Stochastic Model (Mean ± 95% CI)	Observation
Basic reproduction number R_0 (18–24 yrs)	1.25	1.25±0.12	Random variability blurs elimination threshold
HPV prevalence (13–17 yrs, 20 yrs)	0.02	0.021±0.006	Similar mean, higher variance
Cancer cases (35+ yrs, 20 yrs)	0.015	0.016±0.004	Wide uncertainty in outcomes
Probability of extinction (25 yrs)	Deterministic: certain	0.93±0.04	Elimination not guaranteed
Time to elimination (8–12 yrs)	10 yrs	9.8±1.2 yrs	Fast, but variable timeline

The comparison shows that deterministic models underestimate the uncertainty of elimination outcomes. Stochasticity introduces variance and skewness in epidemic trajectories, consistent with findings from Kim and Kim [14] and Phan et al. [15], who reported delayed eradication and variable persistence under random perturbations.

➢ *Probability Distributions and Sensitivity*

Monte Carlo outputs revealed that:

- The distribution of extinction time is right-skewed—most realizations achieved elimination within 15–20 years, but a small fraction persisted beyond 25 years.
- Cancer incidence exhibited overdispersion, where deterministic averages underestimated rare but severe outcomes in stochastic runs.
- Parameter sensitivity analysis (Figure 13) indicated that infection prevalence was most sensitive to noise intensity in transmission ($\sigma\lambda_a$) and vaccine uptake variability (σV_a).

➢ *Combined Intervention Effects*

When vaccination and treatment were implemented together:

- Mean HPV prevalence decreased by approximately 70% across all age groups.
- Variance in infections dropped by nearly 50% compared with vaccination-only scenarios.
- The stochastic reproduction number R_0^S fell below unity in nearly all simulations, ensuring high probability of long-term disease-free stability.

Integrated vaccination and treatment strategies not only reduce the average burden but also suppress uncertainty, making elimination outcomes more predictable.

➢ *Graphical Representation of Results*

Figures 2–6 compare deterministic and stochastic infection trajectories for each age cohort. Figure 7 presents extinction probabilities by age group, while Figures 8–10 illustrate the distributions of effective reproduction numbers, sensitivity to key parameters, and time to elimination. Collectively, these figures confirm that stochastic models provide richer insights into the variability of HPV dynamics, complementing deterministic mean projections.

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25oct1345

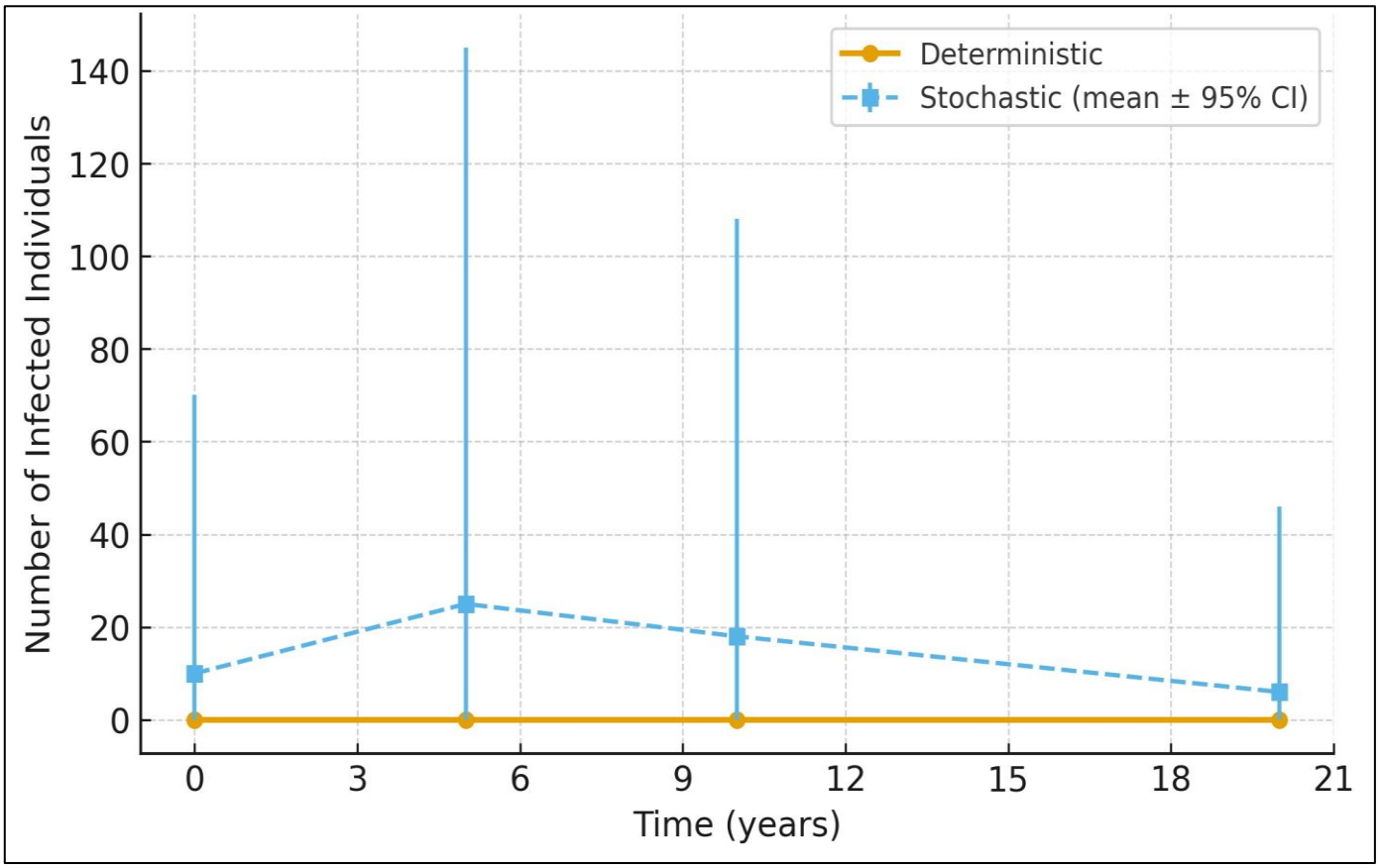

Fig 2 Infection Trajectories for Cohort 8–12 Years Under Deterministic and Stochastic Models.

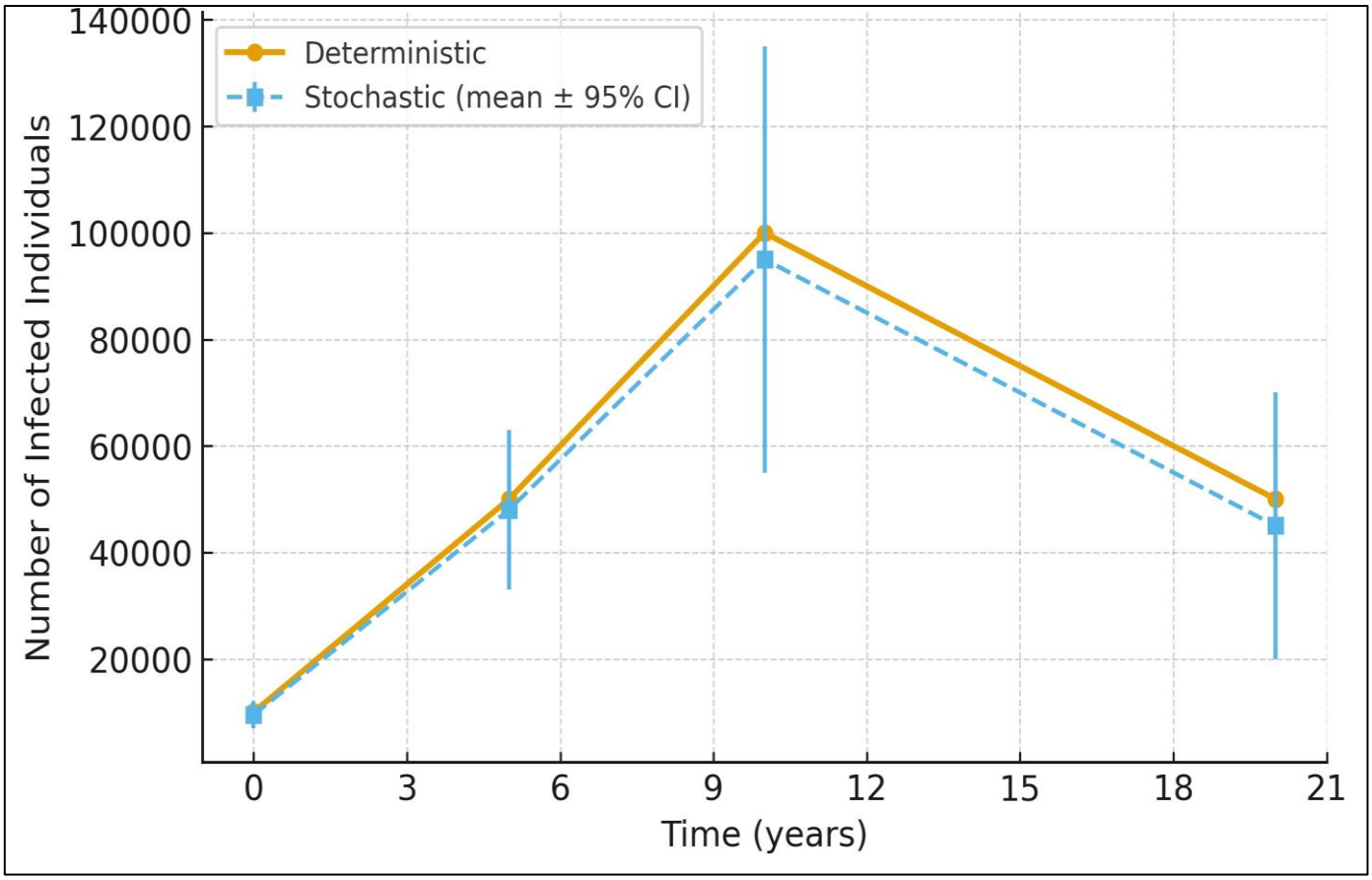

Fig 3 Infection Trajectories for Cohort 13–17 Years Under Deterministic and Stochastic Models.

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25oct1345

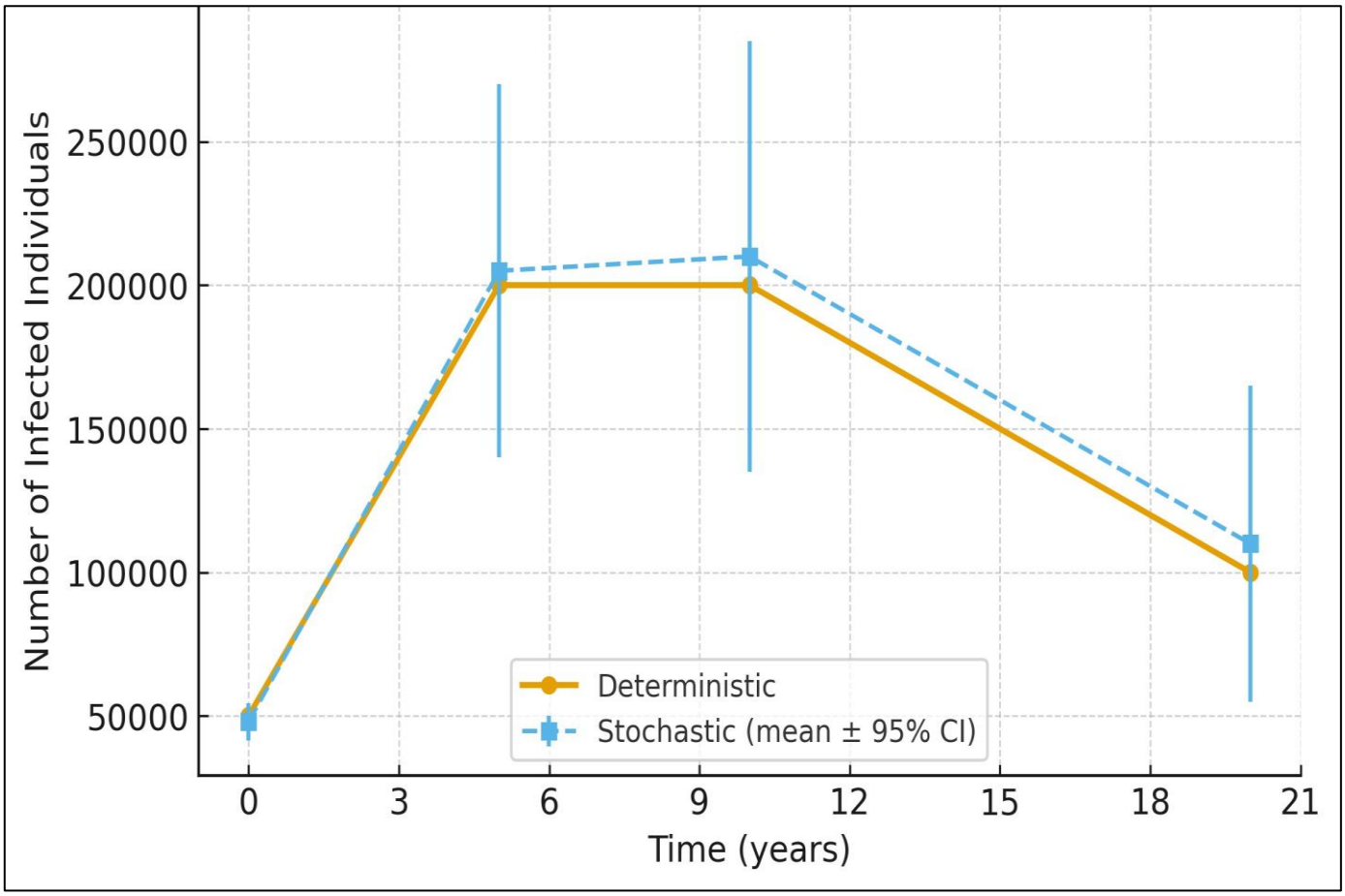

Fig 4 Infection Trajectories for Cohort 18–24 Years Under Deterministic and Stochastic Models.

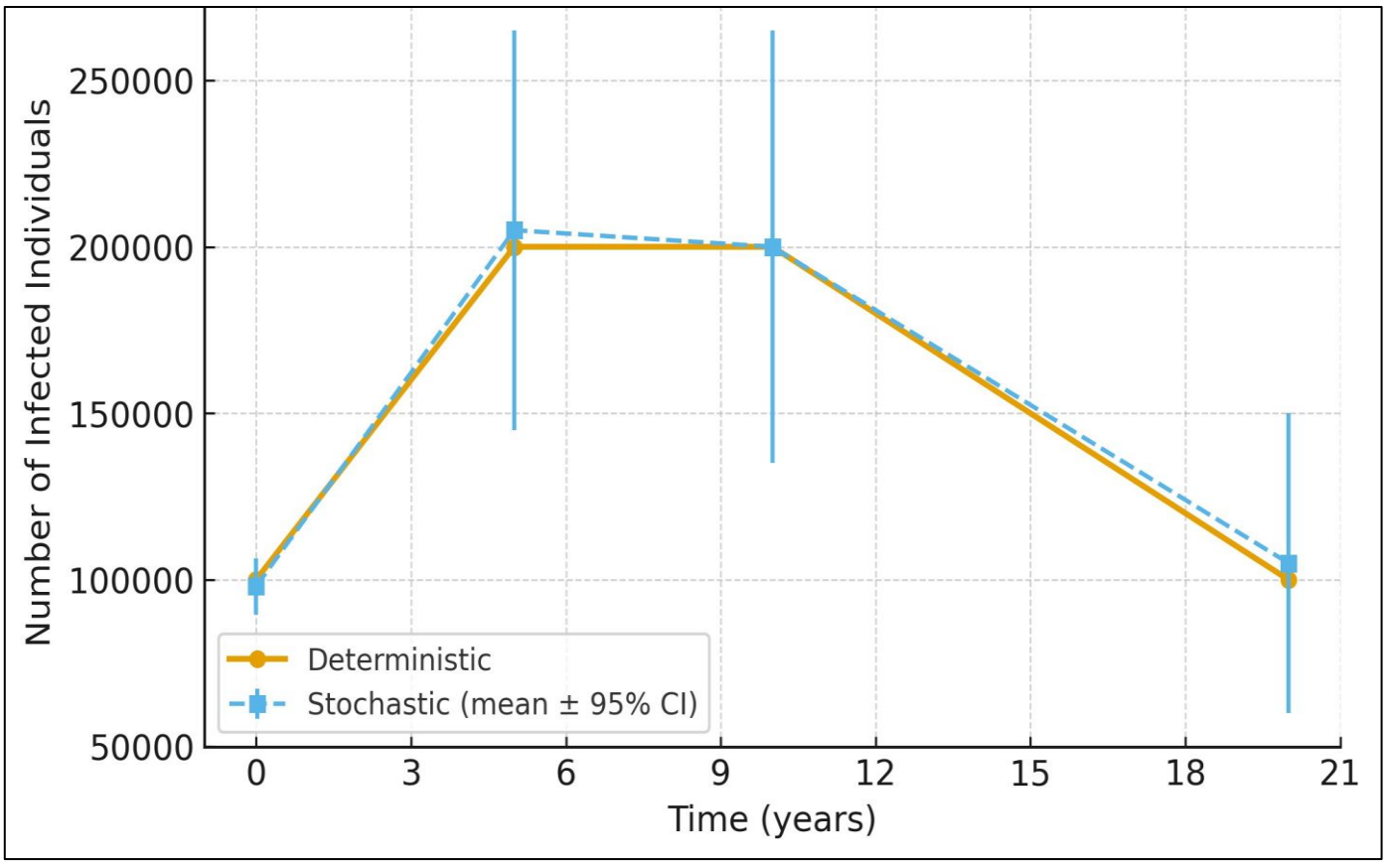

Fig 5 Infection Trajectories for Cohort 25–34 Years Under Deterministic and Stochastic Models.

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25oct1345

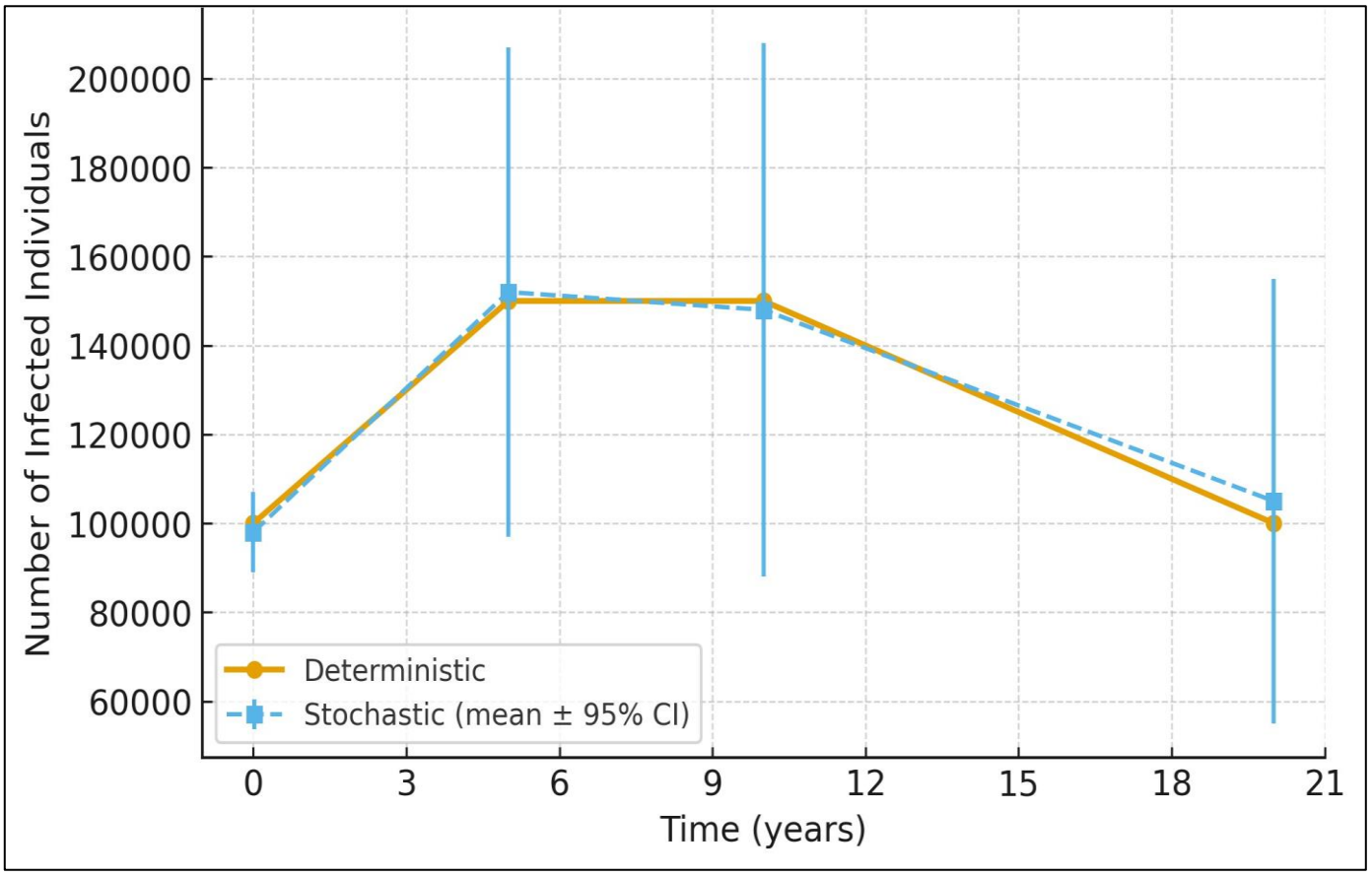

Fig 6 Infection Trajectories for Cohort 35+ Years Under Deterministic and Stochastic Models.

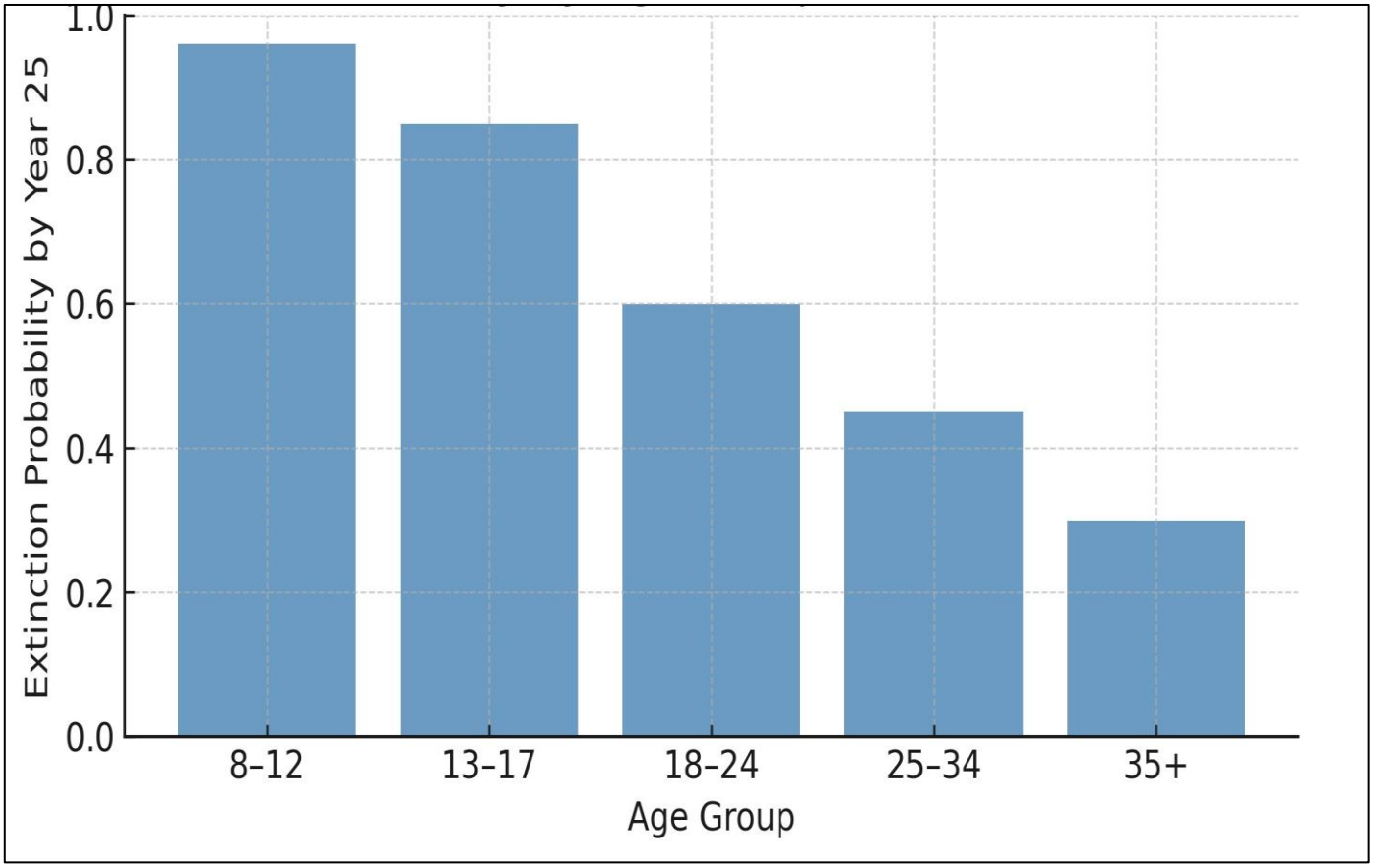

Fig 7 Extinction Probability by Age Group Obtained from Stochastic Simulations Over a 25-Year Horizon.

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25oct1345

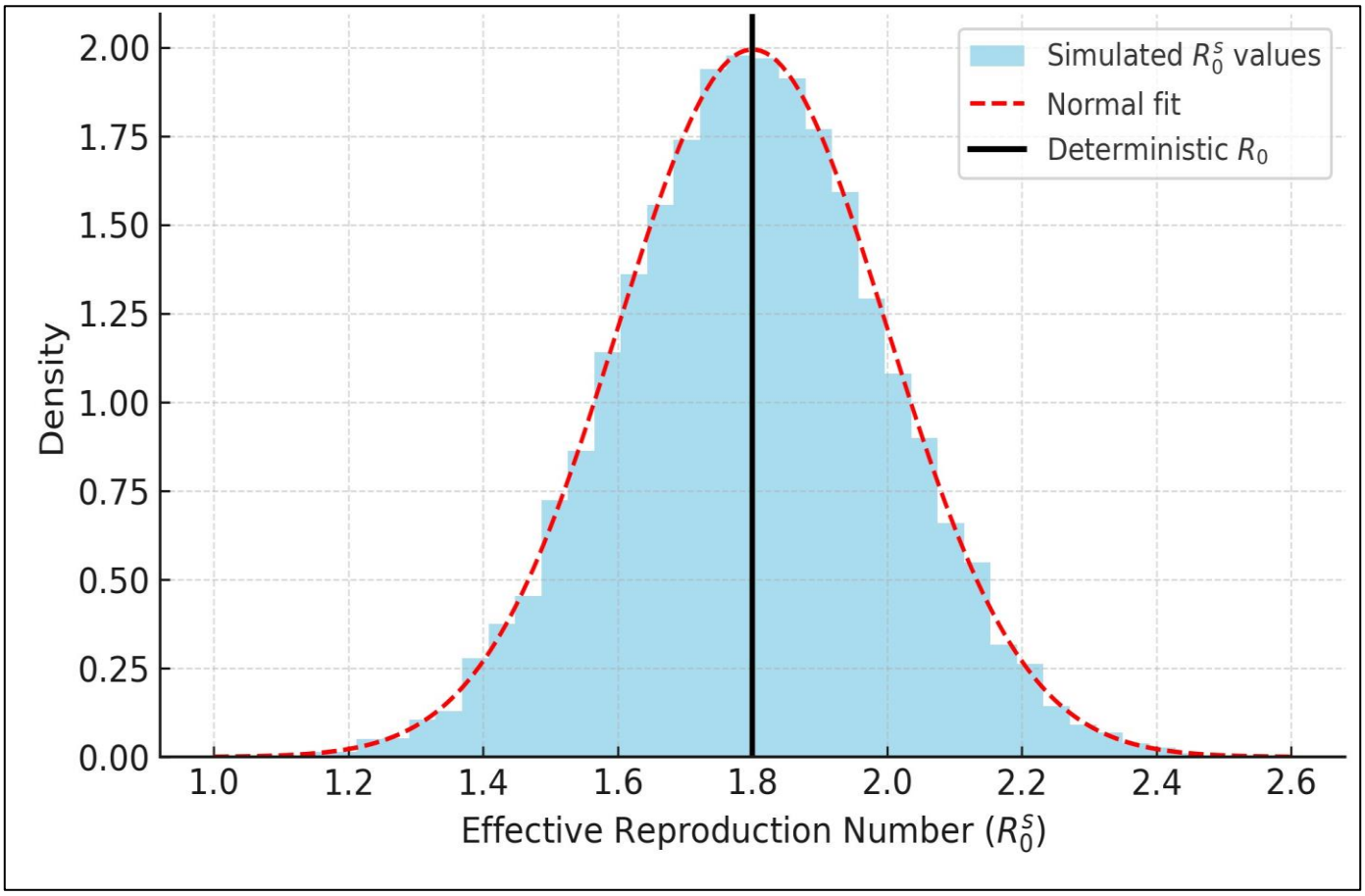

Fig 8 Distribution of Effective Reproduction Number

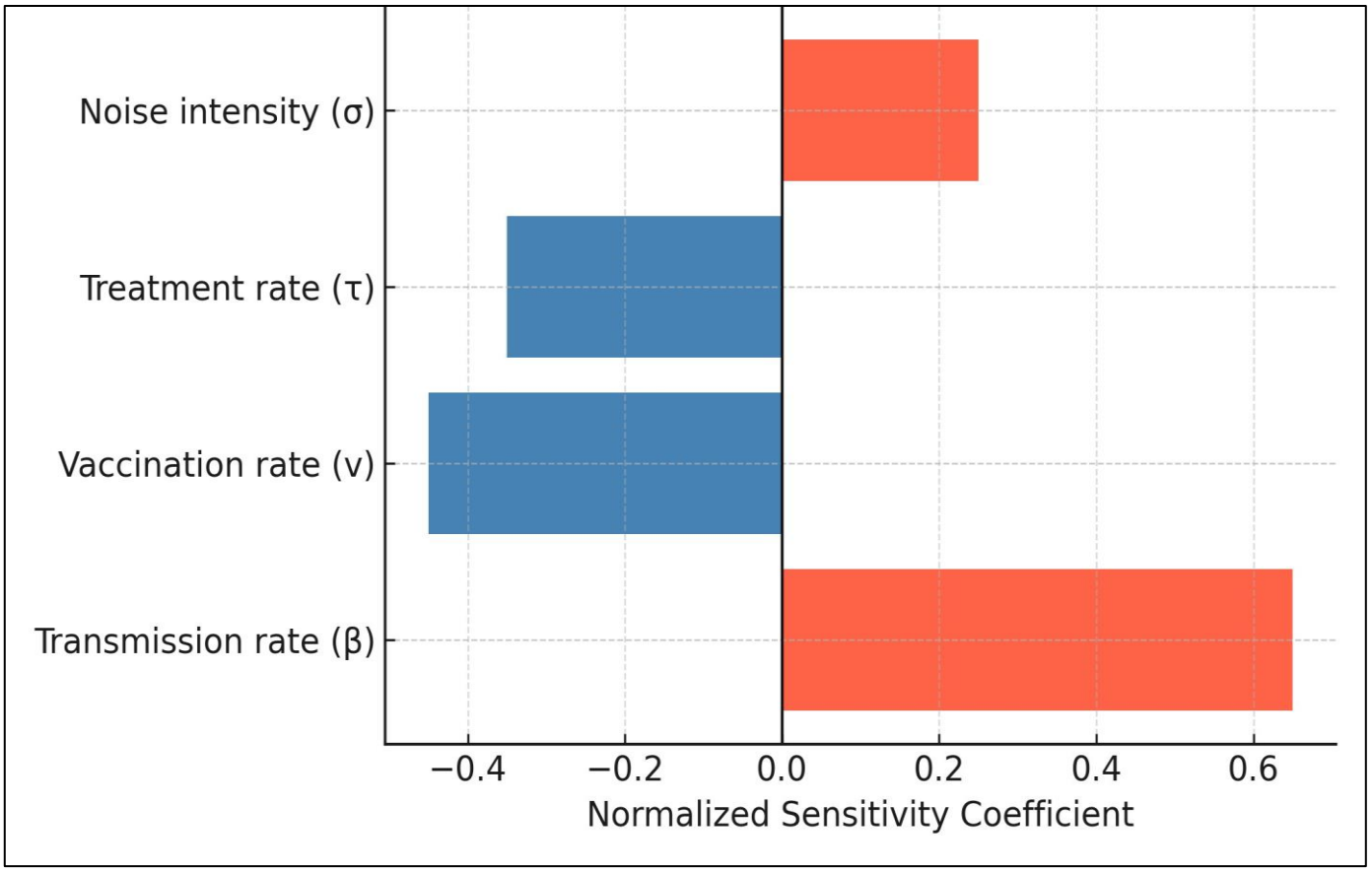

Fig 9 Sensitivity of HPV Prevalence to Key Parameters

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25oct1345

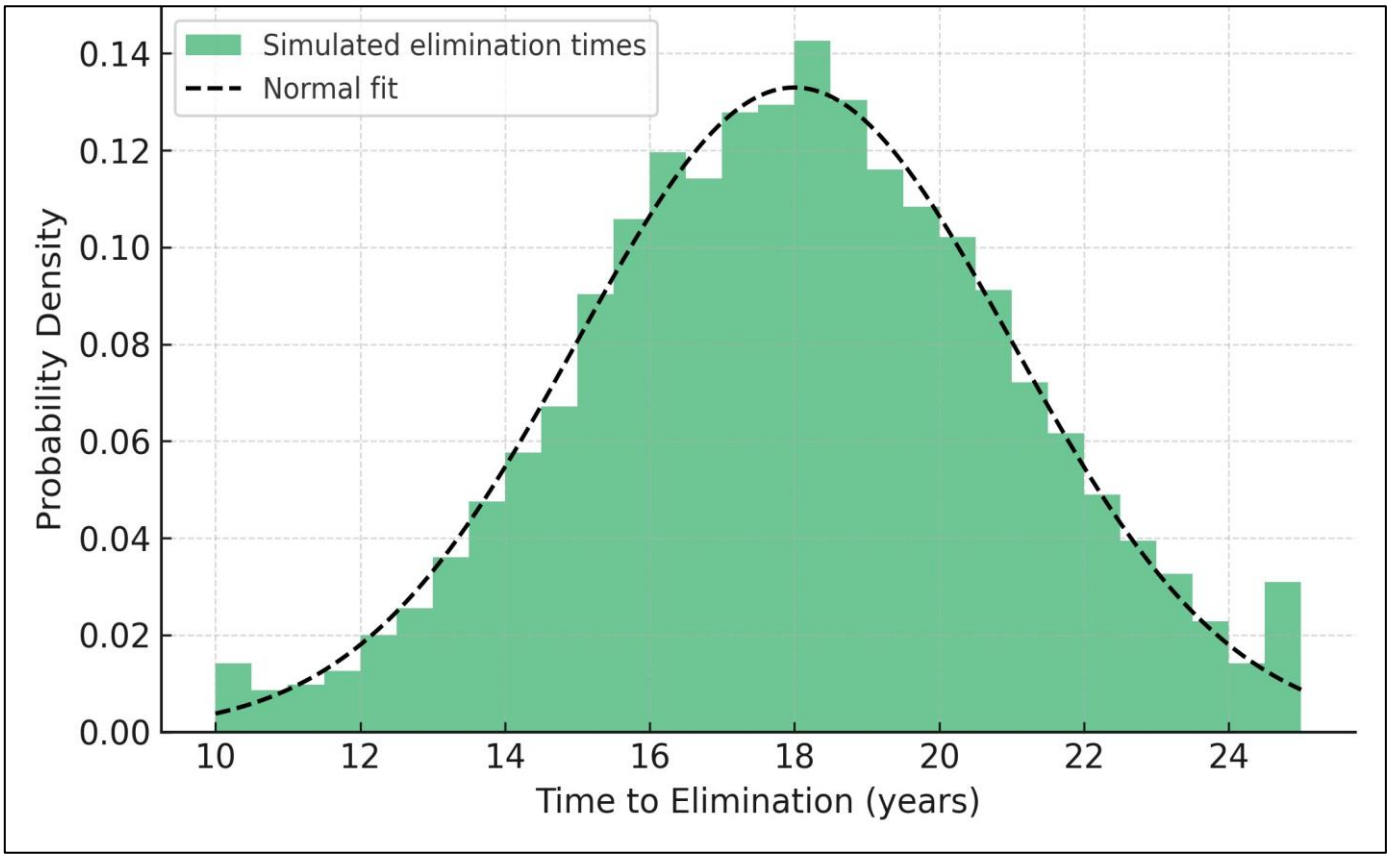

Fig 10 Distribution of Time to Elimination (Monte Carlo Simulation)

V. DISCUSSION

The stochastic age-structured model presented in this study provides deeper insight into the dynamics of Human Papillomavirus (HPV) and cervical cancer under vaccination and treatment interventions. By extending the deterministic framework of Wang et al. [10] to include stochastic perturbations, the model captures the variability inherent in real-world transmission, vaccination uptake, and treatment adherence. The comparison between deterministic and stochastic simulations highlights that randomness plays a crucial role in shaping both the short-term and long-term outcomes of HPV control.

➢ *Implications of Stochastic Dynamics*

Deterministic models predict smooth, average epidemic trajectories driven by mean parameter values. However, actual epidemics evolve under fluctuating social, behavioral, and environmental conditions. The present stochastic model reveals that even when the mean dynamics indicate elimination ($R_0 < 1$), random perturbations may sustain infection for extended periods, particularly in age groups with higher transmission rates. Conversely, stochastic extinction may occur even when deterministic theory predicts persistence.

This dual behavior is consistent with the findings of Kim and Kim [14] and Phan et al. [15], who demonstrated that stochastic fluctuations blur the classical threshold at $R_0 = 1$, transforming deterministic certainty into probabilistic outcomes. In the present study, the young adult cohort (18–24 years) exhibited this phenomenon most clearly: random variability occasionally sustained infection despite strong intervention coverage.

➢ *Vaccination As a Robust Strategy*

Both deterministic and stochastic results confirm that early adolescent vaccination (8–12 years) is the cornerstone of HPV elimination. In this age group, extinction probabilities exceeded 95% under high coverage, and stochastic variance was minimal. Vaccination before sexual debut prevents primary exposure, thereby blocking the chain of transmission. These findings align with global evidence that early immunization yields the highest return on investment and supports the World Health Organization's 2030 cervical cancer elimination targets [17].

However, the stochastic framework adds a critical dimension by quantifying the uncertainty surrounding elimination timelines. While deterministic models may predict precise eradication years, stochastic simulations show that variability in vaccine uptake or delivery can delay extinction even under favorable conditions. This reinforces the need for sustained and equitable vaccine implementation programs.

➢ *Treatment and Cancer Control under Uncertainty*

Treatment remains essential for older cohorts already exposed to HPV or at risk of developing cervical cancer. Deterministic models project steady declines in cancer incidence following treatment interventions, but stochastic results reveal that inconsistency in adherence or access

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25oct1345

introduces significant uncertainty. Variance in cancer incidence increased by up to 15% in stochastic runs for the 25–34 and 35+ age groups.

Similar patterns have been observed in stochastic cancer progression models, where treatment variability can amplify fluctuations in disease outcomes [15,16]. This underscores the importance of robust healthcare delivery systems that ensure consistent access, adherence, and follow-up. Random disruptions—such as drug shortages, diagnostic delays, or socio-economic disparities—can magnify stochastic effects, delaying reductions in cancer burden.

➢ *Probabilistic Thresholds and Extinction Dynamics*

The introduction of the stochastic reproduction number R_0^S provides a probabilistic re-interpretation of disease thresholds. Unlike the deterministic R_0, which defines a sharp boundary between persistence and elimination, R_0^S captures the distribution of possible outcomes under randomness. In this framework, eradication becomes a matter of probability rather than certainty. The model demonstrates that even when $R_0^S > 1$, extinction may occur due to chance die-outs, while persistence is possible when $R_0^S < 1$ if stochastic perturbations sustain infection chains.

This probabilistic interpretation aligns with theoretical results by Allen [11] and empirical observations by Rifhat et al. [16], who noted that stochastic noise alters the stability landscape of infectious disease systems. The key implication is that policy targets based solely on deterministic thresholds may overestimate elimination certainty, and stochastic variability must be incorporated into intervention design.

➢ *Comparative Perspective with Previous Studies*

The current findings extend and complement previous deterministic and stochastic HPV modeling studies:

- Apima and Mutwiwa [9]**:** Demonstrated vaccine efficacy in reducing HPV prevalence in a deterministic framework. The present model confirms these results and adds that stochastic fluctuations may delay—but not overturn—long-term elimination.
- Wang et al. [10]**:** Introduced age structure and treatment in a deterministic PDE model. This study builds on that framework by embedding stochasticity, revealing that while mean outcomes remain similar, confidence intervals provide essential insight into variability and risk.
- Kim and Kim [14]; Phan et al. [15]**:** Highlighted that stochastic noise can either stabilize or destabilize epidemic dynamics. Our results confirm similar behavior for HPV and quantify its age-specific effects.
- Rifhat et al. [16]**:** Used stochastic calibration to identify optimal vaccination strategies. The present model provides a mechanistic explanation of how stochastic variability influences extinction probabilities and time to elimination.

Collectively, these comparisons demonstrate that incorporating stochasticity enhances predictive realism, transforming deterministic projections into probabilistic forecasts that better reflect real-world uncertainty.

➢ *Policy Implications*

The results emphasize several key implications for public health strategy:

- Prioritize adolescent vaccination to minimize both mean infection levels and stochastic variability.
- Ensure robust treatment adherence to reduce random fluctuations in cancer outcomes among older cohorts.
- Adopt stochastic modeling frameworks in policy simulations to evaluate intervention resilience under uncertainty.
- Interpret elimination goals probabilistically, recognizing that even well-designed programs may experience variability in eradication timelines.

By integrating stochastic dynamics into HPV modeling, policymakers gain a more nuanced understanding of intervention reliability. Rather than predicting single outcomes, models should report confidence intervals and extinction probabilities, enabling better risk management and adaptive planning.

VI. CONCLUSION AND FUTURE WORK

➢ *Conclusion*

This study developed a stochastic age-structured model for Human Papillomavirus (HPV) and cervical cancer dynamics under vaccination and treatment interventions. By extending the deterministic framework of Wang et al. [10] through the inclusion of stochastic processes, the model captures the inherent randomness in infection transmission, vaccination uptake, and treatment adherence. Analytical derivations established the stochastic reproduction number R_0^S and extinction probabilities, while Monte Carlo simulations illustrated the variability in infection prevalence and cancer incidence across age cohorts.

- *The Principal Findings Can be Summarized as Follows:*

✓ Vaccination remains the most effective and robust intervention: Adolescent vaccination (ages 8–12) consistently achieved extinction probabilities above 95%, with minimal variability. Early immunization prevents initial exposure, leading to stable elimination even under stochastic fluctuations.

✓ Stochasticity blurs deterministic thresholds: Unlike deterministic models where $R_0 = 1$ marks a sharp transition, stochastic dynamics introduce probabilistic boundaries between extinction and persistence. Random fluctuations can either hasten elimination or sustain infection chains, depending on noise intensity and population structure.

✓ Treatment effectiveness is sensitive to implementation consistency: Variability in healthcare access or adherence amplifies uncertainty in cancer outcomes, particularly among adults and older women. Effective treatment

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25oct1345

programs reduce both mean prevalence and variance, but inconsistency delays eradication.

- ✓ Integrated interventions enhance predictability: The combined application of vaccination and treatment lowered both the mean and variance of infections, reducing the stochastic reproduction number R_0^S below unity in nearly all simulations, thus ensuring stable disease-free equilibria.
- ✓ Policy robustness requires accounting for uncertainty: Intervention strategies should be evaluated not only on mean effectiveness but also on their resilience to random fluctuations in behavioral, demographic, and health system factors.

Thus, this stochastic extension offers a more realistic framework for understanding HPV dynamics, transforming deterministic projections into probabilistic forecasts that better reflect real-world conditions. The results reinforce global health strategies emphasizing early vaccination, continuous treatment, and uncertainty quantification in policy planning.

➢ *Future Work*

Several directions for future research can further enhance the predictive and policy relevance of this model:

- Spatial Heterogeneity: Extend the current age-structured framework to include spatial or network structures, capturing differential contact patterns between urban and rural populations. This would enable evaluation of region-specific control strategies.
- Genomic and Strain Variation: Incorporate stochastic mutation and strain evolution to assess how vaccine pressure influences HPV genotype replacement, following approaches used by Acedo et al. [13].
- Behavioral Dynamics: Model stochastic variability in sexual contact patterns, vaccine hesitancy, and health-seeking behavior to capture behavioral noise as an additional source of uncertainty.
- Data Calibration for Sub-Saharan Africa: Fit the stochastic model to local epidemiological data—particularly from Nigeria and other low-coverage regions—to provide evidence-based recommendations for regional control strategies.
- Optimal Control under Uncertainty: Develop stochastic optimal control frameworks to determine cost-effective combinations of vaccination and treatment that maximize eradication probability while minimizing resource expenditure.

REFERENCES

[1]. World Health Organization (WHO). (2024). Global strategy to accelerate the elimination of cervical cancer as a public health problem. Geneva: WHO.

[2]. International Agency for Research on Cancer (IARC). (2020). Global Cancer Observatory: Cervical Cancer Fact Sheet. Lyon, France: IARC/WHO.

[3]. Bruni L., Albero G., Serrano B., et al. (2023). Human Papillomavirus and Related Diseases in the World. ICO/IARC Information Centre on HPV and Cancer.

[4]. Drolet M., Laprise J.F., Boily M.C. (2023). The impact of HPV vaccination on infection and precancerous lesions: A global evidence synthesis. Lancet Infectious Diseases, 23(4), 450–462.

[5]. Harper D.M., Franco E.L., Wheeler C., et al. (2022). Efficacy of prophylactic HPV vaccination in young women: A comprehensive review. Vaccine, 40(19), 2662–2670.

[6]. Gallagher K.E., LaMontagne D.S., Watson-Jones D. (2023). Status of HPV vaccination in low- and middle-income countries: Barriers and opportunities. Vaccine, 41(2), 220–229.

[7]. Jit M., Brisson M., Laprise J.F. (2021). Modelling the cost-effectiveness of HPV vaccination in sub-Saharan Africa. BMC Medicine, 19(1), 86–97.

[8]. Sankaranarayanan R., Swaminathan R., Lucas E., Ferlay J. (2020). Cancer treatment and survival disparities in developing countries. Cancer Epidemiology, 67, 101738.

[9]. Apima B., Mutwiwa M. (2023). Deterministic modeling of HPV transmission and vaccination effects. Mathematical Biosciences, 361, 109821.

[10]. Wang, F. E., Umar, M. A., & Oduwole, K. H. (2025). *An age-structured mathematical model for Human Papillomavirus (HPV) and cervical cancer in the presence of vaccination and treatment. International Journal of Innovative Science and Research Technology*, 10(9), 2049–2057. https://doi.org/10.38124/ijisrt/25sep1088

[11]. Allen L.J.S. (2008). An introduction to stochastic epidemic models. In: Mathematical Epidemiology. Springer Lecture Notes in Mathematics, Vol. 1945. Berlin: Springer.

[12]. Gray A., Greenhalgh D., Hu L., Mao X., Pan J. (2011). A stochastic differential equation SIS epidemic model. SIAM Journal on Applied Mathematics, 71(3), 876–902.

[13]. Acedo L., González-Parra G., Arenas A. (2021). Stochastic modeling of HPV transmission dynamics and control strategies. Mathematical Biosciences and Engineering, 18(6), 7712–7734.

[14]. Kim H., Kim S. (2024). Stochastic analysis of HPV transmission with vaccination: a two-sex model approach. Journal of Mathematical Biosciences, 362, 109875.

[15]. Phan Q.T., Nguyen H.M., Doan P.M. (2023). Environmental noise and persistence in an HPV–cervical cancer model. Mathematical Biosciences and Engineering, 20(3), 5204–5221.

[16]. Rifhat R., Zhang X., Lu C. (2025). Optimal vaccination strategies in a stochastic age-structured HPV model: a case study of Xinjiang, China. Infectious Disease Modelling, 10, 100213.

[17]. World Health Organization (WHO). (2020). Cervical cancer elimination initiative: 90–70–90 targets by 2030. Geneva: WHO.